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Exponents of Baby Feeding by Physicians ~

The Journal of Pediatrics

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Original Communications

PSYCHOLOGIC ASPECTS OF NUTRITION IN CHILDHOOD*†

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CHICAGO, ILL.

THE development of an intellect superior to that of his fellow creatures, together with the acquisition of a unique manual dexterity made possible by assuming the erect position, has widened man's interests and desires in all directions and has made their fruition possible. It is but natural that in this process eating, one of the fundamental biologic needs and satisfactions, should have received major attention. Primitive man, like the lower animals, had a deep but not a wide interest in food. He ate to live. Monotony did not pall on him because he had not yet experienced the possibilities of a wider choice of form and variety of food. The cow is perfectly contented to eat nothing but grass together with water and a little salt all summer long, and nothing but hay and a little corn all the rest of the year, yet she is never troubled with anorexia. The wild animal has a narrow carnivorous or herbivorous diet as the case may be and does not tire of its sameness. It keeps fit, moreover, by the fact that it is necessary to hunt energetically if carnivorous, and to roam and to be prepared to flee energetically from carnivorous foes if herbivorous. Man is omnivorous, and civilization, so called, has made him increasingly so, not only qualitatively but also quantitatively. He no longer eats merely to live, or to quiet his hunger contractions; eating has assumed a social rôle of vast proportions, and has become one of the major pursuits and pleasures of life. He gathers his foods from all the corners of the earth. An ordinary dinner combines some fifteen different foodstuffs and a standard Thanksgiving orgy at least twenty-five. He has developed the culinary art so that elementary foodstuffs are made to appeal more keenly to his appetite, rather than

*Packard Memorial Lecture, Philadelphia Pediatric Society, Philadelphia, Pa., April 12, 1932.

†From The Children's Memorial Hospital, Chicago.

to the mere satisfaction of hunger. Not only that, his meals are cunningly devised further to appeal to his appetite by a progressive series of approaches from as many different gustatory angles. A six-course dinner has at least six such approaches. When we have finished with soup, meat, vegetables and salad, when our physical needs are fully covered and we are no longer hungry, we still find ourselves agreeably inclined to the additional calories contained in a dessert, and in a cup of coffee with cream and sugar. How far we have departed from the primitive is well illustrated in the Pioneer Meal movement, which asks us to make the sacrifice of eating one simple pioneer meal a week and donating the resulting saving to help the unemployed. One might well wonder whether the word "essen," which the German has arrogated to the more dainty eating of man, might not better be exchanged with the word "fressen," with which he designates the more biologic eating of all other animals than himself. Man no longer hunts for his daily food, nor is hunted, and so physical exertion, except in the case of the laborer, has long ago been replaced by what is known as exercise, a very individual and so a very variable quantity. As a result of all this, man varies amazingly in nutrition and body architecture from age to age, from individual to individual, from family to family, and even from nation to nation. The physical uniformity of the animal no longer exists. This fact alone will probably form the most effective barrier against the universalizing of the nudist cults that are cropping out in European countries, unless, indeed, modernistic art should come to the rescue and convert us to new ideals of beauty.

The introduction of a wide variety of foods into our daily routine has naturally brought with it a wide variety of choice. We satisfy our appetites as well as our hunger. This inevitably leads to preferences, to likes and dislikes, to attitudes as the psychologist might put it. The adult through long experience has learned the necessity for conformity to conventions; the child is still by nature a nonconformist. Whether he shall remain so or not, depends much upon the extent to which the psychologic aspects of eating are recognized and utilized in guiding the child from early infancy to adult life. The child, too, is keenly sensitive to, and largely moulded by, his environment. His wants and desires are still unbridled, and he resents interference with his pleasures. He is swayed and often dominated by emotions, the gratification of which will lead him to refuse to satisfy even such fundamental urges as eating. He is a queer little bundle of psychology, whose mysteries the psychologists and child guiders have not yet fully fathomed. He may have only three primitive instincts in the laboratory, but he has an amazing psychologic repertoire in the home. The home is his natural habitat and there only can he be observed to full advantage. It is for that reason that we pediatricians

feel that the psychologist and the psychiatrist have gaps in their practical knowledge of child behavior that we can help them to fill out. We feel, too, that we are justified in telling our biochemical allies, to whom we owe so much for defining the desiderata of an optimum diet, that a child is not a hopper, or a grist mill, that will grind out whatever is poured into it—but that he is a thinking, preferring, interpreting, emotional little tyrant with strange, often inexplicable and unpredictable vagaries in his little psychology, that, if unheeded, nullify much of their scientific admonitions. It is a far cry from knowing the physical and chemical and caloric requirements of an optimum diet, to the practical application of that knowledge in the home. The laity have acquired an unquenchable thirst for things scientific; science is the thing and they like it straight; and there is no dearth of teachers of all kinds. Much good has come from all this, also much harm. The matter of nutrition in childhood is replete with psychology—not alone the psychology of the laboratory and the books and the lecture room, but even more, that of the common banal mental and emotional reactions to eating in a given environment under a given set of circumstances. Our textbooks are peculiarly silent on the whole subject. It has seemed to me of sufficient importance to warrant my directing your attention to it on this occasion.

Much of what I shall say is very simple. To one unfamiliar with its possibilities it may seem more fitting for the nursery than for a body of scientific men whose study and concern is the child. There is no more important place in all the world than the nursery. It is there that the "twig is bent" that determines how the tree shall incline. Many a child carries through life the ineffaceable imprint of things acquired in the nursery. I should feel inclined to apologize for presenting what may seem so simple a matter were it not for the fact that in my own experience it does not seem to me to be appreciated as fully as it ought to be by pediatricians, general practitioners and nurses; and also for the fact that our more recent allies, the psychologists, psychiatrists, child guiders, mental hygienists, and behaviorists do not seem to grasp the subject as fully and as fruitfully as might be.

The first three months of life offer little of interest from our present standpoint. One still occasionally encounters the erroneous idea, especially in maternity wards and among nurses, where such ideas often linger tenaciously, that a newborn baby has the faculty of choosing one method of taking food as compared with another; specifically stated, that one must not give a newborn baby a bottle, and especially not right after the breast, because the opportunity to choose might lead to an undesirable preference for the bottle. That is too high an estimate to place on the intelligence of a baby in the first month or two of life. The newborn infant will automatically suck anything that is put in its mouth, especially if it is sweet, and as long as the

object holds out any promise. During the first three months or so it will go without choice from breast to bottle and from bottle to breast with no danger of eliminating either at the expense of the other, provided, of course, that the bottle food does not differ in taste so greatly from its natural food that it finds it unacceptable from that angle. It is, of course, well known that a normal newborn baby will suck automatically if something sweet is placed in its mouth, and will make a face and show displeasure at things disagreeably bitter or sour. Our present interest is not in the food but in the container.

From the third month on, choice plays an increasingly prominent rôle. At the fifth month it is commonly difficult but usually possible to switch a baby abruptly from breast to bottle or from bottle to breast, if it has not been accustomed to the alternate method of taking food. The difficulty increases rapidly during succeeding months, and I should place the ninth month as the dividing line beyond which it is not only useless but undesirable to waste time in trying to make the change, under such circumstances, from breast to bottle, the only condition that is likely to arise.

The practical bearing of these observations becomes evident in practice. The pediatrician is at all times concerned about obtaining the maximum amount of breast milk for the baby that needs supplemental feeding. He also knows that the only surely effective method of obtaining that maximum is by the thorough emptying of the breast and that at not too infrequent intervals. The dairy man who can teach us much by very instructive and quite justifiable analogy has long known this in his own field. It is also an established observation that a well-nourished baby will nurse more vigorously, notably in the first month, than will a baby that is receiving an amount of food inadequate to maintain an optimum nursing vigor. It is further well known, or should be, that a baby that is habitually getting an inadequate amount of breast milk, if wholly breast fed, commonly does not react by excessive crying as a hungry baby is assumed to react. In fact, decided inadequacy of breast milk, if prolonged, often leads to a state of tranquility that may merge into a dangerous state of apathy and lack of nursing vigor, a condition easily misinterpreted as one of satiety by a mother or nurse who thinks that hunger is necessarily manifested by crying. There is, then, the twofold problem of obtaining the maximum amount of natural food, and, if this is inadequate to maintain by itself a satisfactory state of nutrition, of insuring that state of nutrition by adding a necessary amount of artificial food. In the first three months of life mixed feeding is safely and best conducted by so-called complemental feeding, i.e., offering the bottle immediately after the breast. This meets the twofold indication of getting all the breast milk possible by the not too infrequent thorough

emptying of the breast, and the assured supplying of an adequate amount of food at each feeding. From this time on, it becomes increasingly safer and better to supplement the breast by substituting as many breast feedings as necessary by bottle feedings, giving then both breasts at each breast feeding for obvious reasons. It is also well to make sure that the milk does not come too easily from the bottle, especially if complemental feeding is continued beyond the safe period in a given case. When it becomes desirable to discontinue breast feeding in the second half year, or thereabouts, it can be done quickly, but gradually enough, by again resorting to complemental feeding. The baby is now old enough to know that the bottle is coming and will not work long at the breast, with its scant promise.

There is a little practical point in infant psychology, along these lines, that often leads to misinterpretation. The baby at the breast commonly falls off to sleep after the conventional, ritualistic twenty minutes that is a part of nursing tradition, commonly much earlier if left to his own better judgment. Long before that time he has obtained all that the breast will yield, except for the small amount that is being made at the time, and that mothers so often erroneously adduce as evidence that the breast has not been emptied. With a full stomach and a most acceptable natural pacifier, he gradually becomes fatigued and tranquilly goes to sleep. In nursing on the bottle, on the other hand, there is no change in quantity delivered, or in effort, until the bottle is suddenly empty. Even though his little stomach is full enough he will naturally assert one of the three fundamental Watsonian instincts, rage, at the sudden interruption. Unfortunately this short, sudden outburst of crying is often interpreted as due to hunger, and the food is increased beyond the necessary, sometimes beyond the desirable or safe, amount. We have here the obverse of the baby that does not cry when habitually underfed on the breast, whose not crying is misinterpreted as satiety.

There are other practical psychologic aspects during the first year. In changing from a bottle to a wet nurse, much precious, even fateful time may be lost in trying to get a marantic infant beyond the workable age to take the breast, when drawing off the milk and giving it in the accustomed manner would make no break in the desirable continuity of feeding. Even more frequent and more important, in this day when wet nurses are rarely employed except in hospitals and under any circumstances have their milk drawn as a rule, is the bearing of all this upon the infant that enters the hospital in a serious state of nutrition or for some serious illness. It is important again to have no unavoidable delay in the carrying out of a feeding schedule. If the baby is on the breast alone, and especially if he is in the second half year of life and the mother still has an abundance of milk, or

even a fair amount, either he ought not to be in the hospital, or if that is insurmountable, then provision should be made for the mother to keep on nursing him at least part of the time until the danger of underfeeding in a crisis is over with. If the baby is on a bottle, he must be kept on a bottle no matter how old he may be. If he drinks only from a cup, much time may be lost by trying to make him take a bottle just because he is a baby. If, again, the older infant will drink neither from a bottle nor from a cup, a not infrequent occurrence if the mother has had, or has thought she had an adequate supply of milk up to that time, then he must be fed with a spoon, or better still, with a medicine dropper tipped with a short length of catheter, or by gavage until he can be inducted into the more usual methods.

Analogous reasoning applies to the food itself. It is unwise "to swap horses while crossing a stream." Babies like and take well what they are accustomed to, and may resist to a dangerous degree in a critical situation a switch from sweet to acid, or acid to sweet milk, or from either to unsweetened tea or barley water. In the barley water days of long ago, probably not even historical to most of you, the last drug I carried with me was saccharine, for obvious reasons.

Inflexible dogmatism about feeding intervals, too, especially our latter-day insistence in some quarters upon an invariable four-hour interval for all babies, does not meet with a favorable psychologic reaction with all of them, in spite of the fact that they adjust themselves to an amazing amount of dogmatism.

The remedy for all this is as simple as it would seem to be obvious. It has long been my custom at my first visit to a newborn baby to stress two points to the mother: to accustom the baby to a bottle from the start and never to let up for a day; and never to force the child to eat, for reasons that I hope to make clear presently. If the baby refuses to take plain water from the bottle, there is, of course, no objection to adding a small amount of cane sugar for a time to a food that already carries 7 per cent of sugar, or about 20 to 25 level teaspoonfuls to the quart. If this should jar the maternal, or grandmaternal, psychology, though it has been my experience that it is rarely jarred about anything if confidence is established by due explanations, then a little saccharine, 1 grain to the quart, can safely be added for a time and gradually lessened. It is commonly, also, well to begin one bottle of milk at the third or fourth month under any circumstances, to insure smoothness of adjustment. If the first attempt to give a bottle is made some time after what I have called the safe period, force will lead nowhere—in fact, it often leads to greater resistance on the part of the baby. The food must, of course, be made to taste approximately like mother's milk. The bottle should then be offered often more in play than by force or with too much

coaxing. A little one-ounce bottle full of milk with a nipple in place used as a plaything, with the hope that he will put it in his mouth perhaps with a little gentle guidance, will turn the trick if anything will. If he once takes a few draws, there will usually be no more trouble. In the meantime the cup, the spoon, the medicine dropper, and in emergency the stomach tube, can all be brought into play.

A pediatrician once told me that he preferred bottle babies to breast-fed babies—that his results were better in the long run. I can see no possible explanation for such results except a failure to recognize and apply the psychologic factors I have outlined. That failure to heed these psychologic factors leads to unpleasant, even serious, results is a matter of common experience, much less frequently now, however, than formerly. I stressed this danger in the following extract from a paper published in 1923:[1]

"One of the commonest experiences in infant feeding is to find a baby that refuses to take a bottle. In my own experience it is the rule that when I see a breast-fed baby for the first time in the second half year of life the mother has discovered, or will soon discover, that her baby resolutely and unchangeably refuses the bottle method of taking food. If she has been a good breast feeder, indeed because she has been a good breast feeder, she will often volunteer the further information that none of her babies would ever take a bottle, as if they were of some peculiar breed with this as a dominant characteristic.

"The following story comes to all of us with such monotonous and melancholy regularity that I have long felt that some far-reaching effort should be made to remedy this condition. A mother who has had until recently an abundance of breast milk finds that her six- or seven-month old baby is no longer gaining, is perhaps even losing in weight. She is finally convinced that her milk supply is inadequate and reluctantly proceeds to supplement it. She consults her neighbor, or rather gets some volunteer advice from that source, or from some baby food concern, or sees her doctor, and decides to give her baby one bottle a day. She is surprised to find that he will not take it. She decides that the time is not yet ripe and puts it off a few weeks, stuffing in the meantime to increase her own milk. Renewed trial leads to the same result. Now thoroughly alarmed she spends hours trying to force him to take it; again she coaxes and teases and tries to cajole him into taking it, even hiding the bottle under her waist. Forceful measures seem only to whet his determination. He feels now not only starved but abused for no good reason and he deeply resents it. The sympathetic neighbor offers a solution, the Hygeia bottle with its dome shape and tapering nipple fantastically resembling his mother's breast! He flatly refuses to see the resemblance.

Frantically she takes to the cup only to find that most of the milk runs down his neck, if it leaves the cup at all. The spoon gives little better results, but it too is very inadequate if he has not been accustomed to taking spoon food for some months past and this is usually the case. In the panic that seizes her as she sees her baby losing in weight and refusing all other food the remaining breast milk leaves rapidly. Heroic measures must now be taken! She withholds the breast altogether; he must be starved into taking the bottle. She now makes the alarming discovery that it is not food that he objects to but the receptacle from which it is offered, not the contents but the method. He would refuse his mother's milk from a bottle as decisively as he does cow's milk. He would take cow's milk as gladly from his mother's breast as he would her own. He is now losing rapidly and plainly shows it even without scales. The last food supply is now gone, her breasts are empty. She rushes to her doctor. A few more days of floundering, a still greater loss in weight. She goes to the specialist, only to find that he too knows no royal road to remedy the condition; that he knows no way to change a baby's psychology of habit; that at best he can bring a little more intelligence and a little more experience to the problem, but that its solution is a makeshift. The mother whose baby had been all that could be desired and whose breasts were the envy of her neighbor whose pale, thin, scrawny, crying baby had been struggling along on calf's food, suddenly realizes that that other baby has outdistanced hers in the second semester and is maintaining a steady and serene lead. She soon learns, further, that all the devices she and the specialist can invent as a rule will not make the baby take as much milk as is desirable, and that he will remain undernourished for some time to come. If, as so often happens, there is combined with this a disinclination to take spoon food because it was begun too late, the matter assumes even greater importance.

"The situation may indeed become even acutely fateful. I remember vividly an infant of about fourteen months who had nursed an abundant breast for a year. Attempts to get him to take a bottle, or cup, or spoon food at that late period were unsuccessful. The mother now proceeded to starve him into it by withholding the breast. He refused all food and took practically no water. When I saw him in consultation several days later he was in a coma with a marked hyperpnea, would no longer take the now empty breast, and died a few hours later.

"Is all this a fanciful picture I have drawn? To me it is one of the most real, perennially recurring, and pathetic pictures I know. No wonder that the same mother with a second baby will say: 'What's the use of having lots of milk of my own? Look at my first baby and

then at my neighbor's. I'm going to give this baby a bottle only from the start.'" That this condition is less frequent now is doubtless due to the twofold fact that few babies today are allowed to escape at least some medical supervision, either directly or through the medium of the press, and that artificial feeding has become so simple and safe that it is no longer put off to a dangerous period.

As I have intimated, there is an analogous psychologic and an analogous chronologic setting to the giving of other foods during the first year. If spoon food, hand food, and coarser nonsieved foods are not introduced at approximately the right time, much trouble may be experienced in adjusting a child to the conventional diet of a later period. It is interesting to note that grandmother and the baby knew this long before most of us did. It is my own practice to begin spoon food, cereal from a spoon, at the third month. That is my present stopping place in the long and gradual descent from a much more advanced age. I have no quarrel whatever, on physiologic grounds, with those who begin cereal feeding earlier—that babies knew how to digest large quantities of starch in earliest infancy long before we did, and that it can fairly easily and safely and effectively be pushed down to the level of the swallowing reflex has been amply demonstrated in the feeding of thick cereals for pylorospasm. To give spoon food during the first two months on psychologic grounds seems to me quite unnecessary and is, at best, a sort of acrobatic stunt that lacks pedagogic warrant. There is commonly a little difficulty in giving spoon food when first offered, even at the third month. Force and undue coaxing again play no part. The addition of some milk from the bottle to insure a familiar and acceptable taste and the free use of a sprinkling of cane sugar at the start and as long as necessary soon overcome the initial difficulty. To yield to this resistance by putting the cereal in the bottle, as mothers are prone to do if not cautioned, of course misses the point and purpose of the whole procedure and makes it all the more difficult at a later period.

At about the fifth month sieved vegetables in the form of a purée can commonly with a little patience be introduced as further spoon food. There is a nutritional advantage in cooking a number of vegetables together with only enough water so that it can be incorporated in a final dish of the consistency of a porridge. Salt adds to the acceptability; even sugar may be added for a time if necessary. Vegetables must not, however, be given in the form of purées too long. If the introduction of coarser foods is delayed far beyond this period, almost surely if delayed to the end of the first year, it is often extremely difficult to get a child to take anything that is not finely divided. He will commonly at this late period take the coarser food in his mouth, squeeze out the juice, and after a time, often an incred-

ibly long time, will return the rest, often the accumulation of several mouthfuls. The older child, even the adult who simply cannot swallow a pill, can doubtless sympathize with him. The disinclination, or inability, to take coarser adult table food has large possibilities for maladjustment, from both a nutrition and a behavior standpoint, as every practicing pediatrician knows. All of this occurs much more commonly in the so-called better homes, where food is prepared most meticulously and reaches its acme in the hands of the infant nurse who has a very important baby on her hands whom she endeavors to shield against any possible digestive upsets. Parenthetically, I think I might say psychologically, in this connection, there is nothing better than a good infant nurse, and in all medical contacts there is nothing worse than a bad one who has been jelled by age and narrow experience into thinking that she knows more than the doctor and leads the mother to believe it.

For all of these reasons it is my own practice to make several fundamental changes somewhere between the seventh and ninth months of life—not later. These consist in the introduction of coarser foods, as already indicated, and of a larger variety of food so as to make the transition to a later conventional diet more sure and unbroken. The purée is continued for physiologic reasons, but at least half of the time vegetables are offered merely mashed with a fork for psychologic reasons. This change extends also to such things as milk toast, coarse or dry cereals, puddings, scrambled eggs, very ripe bananas and stewed fruits, hand food such as dry toast, crackers or zwieback, and even, on occasions, to scraped or finely cut up beef. The indication for a larger variety of food is clear cut to the experienced pediatrician. It is also clear that there should be established a happy medium between monotony and variety of food. A baby does not tire of monotony in diet as does the adult. For many months he takes the breast or the bottle alone as contentedly as the cow does grass. If as a result, a given vegetable purée or a single cereal is continued too long, it becomes increasingly difficult to change to other vegetables or combinations of vegetables, or cereals. On the other hand, too great variety inevitably leads to preference and rejection with resulting conflict between what seems desirable on the part of donor and recipient. If these points are kept in mind, it is usually a very simple matter, barring adverse environmental influences, to keep the child interested in all new foods. The younger the infant of this age, the less long continued the monotony of diet, the easier and surer and more natural the process. Force again plays no part in the whole procedure, and gentle coaxing only to the point of overcoming a passing resistance.

The transition from bottle to cup is not without both nutritional and psychologic interest. Nearly all of the books, medical and otherwise, advocate the discontinuance of the bottle at the beginning of the second year. Dogmatism here may again lead to trouble. In my own observation the abrupt, or even the gradual complete change from the bottle to the cup at this early period leads quite regularly to a reduction in the amount of milk taken, commonly a very decided reduction. There is, on the other hand, a very real danger that if the bottle alone is continued too long it will be increasingly hard to get the child to take milk from a cup. As an extreme example, I know of a young woman of eighteen years who simply could not drink milk except from a bottle. It has always seemed to me that a middle course offered the best solution. The baby should be taught to take milk from a cup not later than the end of the first year, preferably even earlier. If he takes it as well, and as much from a cup as from a bottle, well and good. If not, then I think it wise to continue a morning and (or) bed-time bottle into the second and even the third year, letting him take what milk he will from a cup and in his other food at the other meal, or meals. Custards and junket also suggest themselves. In this manner the way is paved for cup feeding alone at a later time when he may be more reasonable, and at the same time it assures at least a pint of milk a day, in which I am more interested during this period than I am in the dogmatic "Quart, or more, a day" of our laboratory colleagues, lay writers, dietitians, and milk producers. The early morning bottle is often a great convenience in the domestic prebreakfast scramble, and the evening bottle is a good natural soporific for the baby to go to sleep on after he is tucked in bed, not before. There is, moreover, a definite advantage in the ready ability to take milk or other liquids from a bottle, to the seriously sick child in the second or third year of life, who ought to be disturbed as little as possible.

There is a bit of child psychology about the use of our so-called protective foods. Some babies do not like orange juice, not a few vomit after taking it, not infrequently also the next feeding. If this latter is on an allergic or other intractable basis, then it must, of course, be stopped and other fruit or vegetable juices substituted. The mere dislike often disappears when the orange juice is put in the milk, a procedure quite in line with the acid thought of the day, though the grandmother may be skeptical about the moderns. Much more can be said about cod liver oil, which most of us doubtless still prefer to viosterol. We are prone to think of acceptability of, and tolerance for, food in terms of our own reactions. In spite of the common view that babies simply cannot possibly like cod liver oil, they do. There are exceptions, as in nearly all matters medical, but

with proper technic these are few. It must, however, be begun early, preferably in the first month, and must be given daily without a let-up. In that early period babies will take many things that they would refuse later, and having once acquired the taste it remains as fixed as that of the adult for tobacco, or whisky, or olives, or other initially unpleasant things. It is by no means uncommon to have a mother say that her sick child refuses all food except his cod liver oil. There is a current idea that seems to me quite ill founded that cod liver oil must be discontinued during the summer months, both because it is "too heavy," whatever that may be, and also because it is unnecessary because of the greater abundance of ultraviolet rays. If cod liver oil is stopped for some months, it may be almost impossible to give it again during the winter months. If one shares the idea that it is "too heavy" for warm weather, then one can still keep up the habit by giving only a small amount daily. If the baby objects too seriously to cod liver oil, then it should be discontinued and viosterol, or radiation, or irradiated foods should be substituted. To put it in the milk probably makes not a little stick to the bottle and may extend the dislike to the bottle food itself and is hard on the dish washer. The same thing applies in a less degree to giving it in fruit juices. Forcing the baby, or young child, to take the oil from a spoon too may start a conditional reflex against all spoon food.

Maternal psychology, too, has an important bearing on the baby's nutrition during this early period. A very large number of babies are now born in hospitals, and the care of the baby is bathed in an atmosphere of ritualism that makes a deep and often ineffaceable impression on the mother. All is well in the hospital during the only vacation some mothers have, but the mother is already worried about whether she will be able to meet the exactions of such ritualistic care when the burden is thrust upon her at home. She is likely to feel that she must learn the rules instead of using her head and her common sense. She is afraid of the consequences if she does not learn the exact manifold usages of the cotton-wrapped toothpicks and the mystic contents of the boric acid jar. Because of the well-known effect of nervousness and worry on the supply of milk, something, again, quite familiar to the dairyman, the mother is often hedged about by a conspiracy of silence, which she quickly senses, and that is far worse in its effect than would be an evident frankness about any minor, remediable, trouble with the baby or with herself. As a result of all these things it is a very frequent experience to have whatever milk there is, and even when there is an abundance, become rapidly lessened and even lost almost in a day when the mother goes home from the hospital. I know of no place in medicine in which a frank, open, personal, sympathetic, common-sense interchange of ideas will

do more good than in just this situation. We are told that there is a hospital in Germany of which it is traditional that the mothers of all babies born there can nurse them, and so they do it. If this is true there must be a fault in our system, or else a difference in the breed, or both.

The effect of a nervous mother and a nervous environment in the home both on the child's disposition and on his nutritional progress is well known to every practicing pediatrician. An analogous condition presents itself in an infant ward in a hospital or in an orphan asylum. It has long been observed that nearly every young infant that enters an infant ward with normal or fairly normal digestion, within a few days has a marked increase in the number of stools. It is also obvious that an infant ward presents a striking contrast to a normal, peaceful home in the number of disturbances to which the baby is subjected. What with regular hours of feeding, the daily bath, the frequent changing, the many temperature determinations at fixed hours, the several examinations a day by interns, attending physicians and otologist, the crying of other babies, the talking of attendants, the changing of nurses, the resulting broken sleep and rest, the new arrival has little of the peace and quiet that is so essential to his physical and psychic well-being. In a convincing experiment Meyer and Jahr[2] have demonstrated that there is a cause and effect relationship between this psychic trauma, on the one hand, and the initial diarrhea and its continuance if once established, on the other hand. By putting one infant, later two, in the sole charge of a competent nurse, day and night, at first in the nurses' home, later in the infant ward, they were able with every one of thirty infants both to obviate wholly the initial diarrhea and to bring an established diarrhea to a speedy termination without change of food. They believe that the many psychic disturbances of an infant ward lower the threshold of tolerance to the invasion of the new and manifold bacterial flora to which the child is subjected in the hospital as compared with the home, and that by special nursing the threshold is maintained at a sufficiently high level to prevent digestive disturbances and to cure them if present. While economic considerations offer a serious obstacle to carrying out a special nurse régime for all infants in hospital wards, there are, at least, many ways in which the underlying principles can be utilized to advantage.

Other psychologic aspects of nutrition in infancy are so inseparably interwoven with those of later childhood that they are best considered together. These can all be grouped around one word, anorexia. In my earlier days anorexia was a good dictionary word, something like oliguria or adiadokokinesis, designating a condition characterized by a disinclination, or even refusal, to eat. We were, of course, well aware

of the fact that children seriously ill did not care to eat. We also knew that there was a condition known as *anorexia nervosa*, a perplexing clinical entity described in some textbooks or magazine articles, but rarely found in practice. There was much speculation as to the pathogenesis. Several things were evident. These victims of *anorexia nervosa* presented no ascertainable physical cause for their refusal to eat. They were said to be nervous or neurotic, came from nervous or neurotic homes, were high-strung, unmanageable, would not eat at home but sometimes would if taken to a hospital; sometimes they got to so alarming a state that gavage seemed necessary to maintain life; and, if improved in the hospital, would quite regularly relapse when they returned home. I cannot recall ever having had a child in those days upon whom I could pin the diagnosis under that name. I remember hearing of one in the practice of a pediatric colleague. I am still a little confused in my own mind as to whether these very isolated cases were *sui generis*, or were merely the pioneers, or outposts, of the army of anorexia that has since descended upon us. I am inclined to think the latter is the true explanation. Under any circumstances, anorexia in children has become the pediatric meal ticket in the last decade or two. A very prominent pediatrician once told me that he had paid for his house with anorexia, and it is no small house! Lucas and Pryor[3] have called attention to "the so-called 'hunger strike' commonly encountered in the practice of pediatrics." About twelve years ago I wrote for Abt's *Pediatrics:*[4] "The child that will not eat represents probably 10 to 20 per cent of children in private practice between the second and the fifth year inclusive, with a lesser incidence in the first year." About two years ago I wrote:[5] "Now anorexia is not an occasional occurrence, an isolated phenomenon in childhood; it is the rule in that very stratum of society in which mothers are lying awake nights planning a Gospel diet and the most effective way of administering it. The lowest estimate of its incidence in private pediatric practice is 50 per cent. A scientific survey in a university neighborhood and a pediatrician with a large office practice in a prosperous suburb have placed it at 81 and 85 per cent respectively."

The first reaction to such evidence may be, not unnaturally: "What of it? What harm does it do?" To my mind there is a good deal "of it." In the first place the condition is so fantastically unnatural, so amazing a biologic phenomenon, the majority of the young of any animal refusing to eat normally, that it at once presents a challenge and demands an explanation. If 50 per cent of a farmer's pigs refused to eat unless forced to do so, he would at once know that there was something fundamentally and seriously wrong and that there was a priori only one cause. In my early days on the farm we should, at

once, have known that hog cholera had at last included us in its path. The situation is all the more startling when we add to it the fact that, unlike the pigs, these children present no evidence of physical disease as a cause. In the second place, these children are commonly malnourished, either quantitatively or qualitatively, more often both. Paradoxically, not a few of them that are said to "eat nothing at all" present the appearance of normal health because they are literally forced to eat. In the third place the child that will not eat is *ipso facto* a problem, a maladjustment that inevitably ramifies far beyond nutritional confines. Anorexia in children, with all the temperamental and environmental forces that surround it, is easily one of the major factors, if not the greatest factor, in the pathogenesis of the full-blown psychiatric problem.

In the etiology of so widespread a condition at a time in life when it should be least in evidence there would seem, at once, to be only one causative factor as compared with which all others would sink into insignificance. Various attempts have been made to explain it on one physical basis or another, or on a number of such factors, endogenous, or exogenous, in spite of the fact that these children present no evidence of illness in the ordinary sense. Hypochlorhydria has been found and suggested as a factor. It would seem far easier to believe that effect is here confused with cause than to believe that the majority of children have this condition. The same would be true of the attempt to find an explanation in type of body build. Lucas and Pryor[5] have recently reported that 82 per cent of 110 children who were on a "hunger strike" presented the linear type of body build in contrast to the remaining 18 per cent, who presented the lateral, or intermediate type. It is interesting to note that in the same journal, Rosenberg[6] presented several "before and after taking" photographs, from which it would appear that the linear type had been changed to a lateral type by the use of a special diet. The excessive use of milk has been assigned an important rôle. Again, in the same journal, Siemsen[7] has produced experimental evidence that speaks against this assumption. One fraction of vitamin B has recently been incriminated from laboratory data on experimental animals. These data are unquestioned. There is, however, a huge gap, that no analogy can span, between the laboratory animal with its artificially and unnaturally restricted diet that does not with certainty exclude other factors, and the normal child in a normal environment with normal food provided by a mother who is rather intelligently scouring the literature and the shops for the latest in vitamins. The factors that enter into child nutrition are so complex, so quixotic at times, that it is quite unsafe to make deductions from human ex-

perimentation in this field. For several months there has been in my closet a big package of a most potent vitamin B containing substance from one of the leading pharmaceutical houses for me to try out as to its effect on appetite in children. I have not tried it out because I would not believe my own observations and conclusions. A change in environment, the mere fact that something is being done, a sudden inexplicable shift in the child's attitude may change the whole picture so that deductions as to a single factor may be wholly misleading. This brings us to the crux of the matter.

In the absence of any one ascertainable physical cause, or combination of causes, we are forced to believe that the essential factor is psychologic in nature. There is abundance of evidence to support this view. It is a priori unthinkable that it is anything else, it does not occur among adults in the same environment, it is perfectly obvious in practice, and there is much objective evidence that seems irrefutable, strange as it may seem. This condition occurs only in the home unless a similar environment is created elsewhere. I have often made the statement that it does not occur in the orphan asylum, or the convalescent ward, or the summer camp. This statement was questioned at a meeting of a committee of the White House Conference. Dr. Alfred Hess went home from that meeting and wrote me a few days later: "I find that among our 315 children in the orphan asylum there is only one with a notably bad appetite." A closer analysis of that case might be interesting. To one familiar with the racial aspects of this problem it is doubly significant that all of these 315 children are Jewish. In an orphan asylum with which I have a consulting connection I asked the attending physician about the situation there. Her impression was that they all ate well except in one room in which the matron "fussed a lot about their eating." I happened in one Sunday while they were all at dinner in one large room. It was a real treat to one coming in from practice. I asked the matron if they all ate well. Her reply was: "Yes, unless they are sick. Come around in fifteen minutes and see them get their second helping. And this is Sunday so they will have ice cream and cake today, too." Equally, or even more, convincing is the fact that anorexia is cured rapidly and almost unfailingly when the child is sent away from home to an orphan asylum, or a hospital ward, with no determining change in food, certainly not for the better. If the mother, or the nurse, is allowed to accompany the child, or even to visit him, there will be no such assured therapeutic result. It would seem impossible to harmonize these observations with a causal hypochlorhydria, body build, vitamin B deficiency, or, indeed, any food inadequacy, occurring in a majority of the children brought up in otherwise excellent homes.

How can one explain anorexia on a psychologic basis? Only one who has lived and practiced through the history of infant feeding and child care in this country in the last twenty-five or thirty years can fully grasp it first hand. I do not believe the situation is as acute in other countries for reasons that may be deduced from its genesis. I shall quote freely from what I have said on a former occasion:[5] "With the rise in knowledge of scientific infant feeding about four or five decades ago, replacing the empiric and inefficient methods that preceded, there naturally followed a long period of meticulous adaptation of this knowledge to the practical feeding of the infant. With the knowledge and the materials then at hand it seemed necessary to construct complicated formulas, to have regular feeding intervals, and to regulate with great precision the amount of food per day and per feeding. We were naturally enough obsessed, and I think rightly so under the then existing circumstances, with the danger of overfeeding, in general, both as to each feeding and as to each food element, and we were precisely concerned with intervals, percentages, quantities, and stools. A more or less rigid system was thus foisted on a psychic individual with a psychology of his own with many strange little quirks and twists, and he normally resented the resulting inability to assert his individuality. He had his likes and dislikes, his whims, temporary indispositions and disinclinations to taking food of a certain kind, at a certain time, of a certain externally predetermined and inflexible amount, and he naturally and normally rebelled against the extraneously, arbitrarily imposed authority that the mother dutifully inflicted and carried out to the letter. While this concerned chiefly the infant, much was carried over into later childhood."

"The problem of anorexia, especially in the older child, has become far more acute in recent times, largely traceable, I think, to the campaign of weighing, measuring and examining all children that came as an aftermath of the World War, and to the further avalanche of information and admonition to the laity that followed from a hundred sources. Untold good was accomplished in bringing to light and removing or lessening such causes of undernourishment as adenoids, infected tonsils, carious teeth, improper food, anemia, heart trouble, and lack of rest, fresh air and sunshine; it has also done immeasurable harm as the big factor in bringing about the condition that I am discussing. Parents have been led to think that their children must be standardized, that they must conform in weight and height to an assumed norm as determined by averages expressed in tables of weights and measures that are now found in all baby books, most nurseries, and on the walls of the offices of nearly all physicians who care for children, except on mine. A few pounds above, even more a few

pounds below the figures, call for standardization. The mother's eyes are fixed on the scales as the final arbiter of nutrition; color, tissue turgor, activity, endurance, race, individual and familial body architecture are lost sight of as the real measures of nutrition . . . . . Bombarded on all sides by lay writers and radio talkers; by books on infant feeding, child nutrition, hygiene, training and care; by dietitians and nutrition investigators; by promulgators of child training and parent education; by parent teachers' associations; and by child health, mental hygiene, and afternoon tea conferences, kaffee-klatches and bridge parties, the mother carries on the struggle of trying scientifically to standardize an unstandardizable product, finally handicapped by the feeling that she is a failure with an undernourished, non-eating, and rebellious problem child on her hands. As my good friend, Dr. Lippman of St. Louis, once said to me: "With all our weighing and measuring, and all our rules and regulations as to when, where, what and how much to feed children we have succeeded in doing just one thing—we have taken their appetites away."

When we look more closely into the immediate pathogenesis of this psychic anorexia, it seems pretty clearly and simply to lie in an abnormal stimulation of two normal human traits by an external irritant. The human being, innately, has a generous fund of negativism freely shared with the higher lower animals. It manifests itself as a rebellion against externally imposed authority, especially if it seems unnecessary, unjust, irritating, nagging, or arbitrary, or if even a mere curtailment of personal liberty that may involve no discomfort or hardship whatever, such as is found, for example, in prohibition, or in traffic regulations. We go even further, we rebel against virtue and greatness if we hear too much of it, as all good presidents have learned. The Athenian voted to exile Aristides because he was tired of hearing him called "the Just." Negativism was doubtless a primitive virtue that still appears, *ad maximum*, though normally and innocently, in the child that has not yet been mellowed by the inhibitions of convention.

The second normal human trait is a desire for the spotlight; to be distinctive whether good, bad, unusual, or queer; to wield power over others on the one hand, or to elicit sympathy on the other. How well this trait is developed in children who have as yet no inhibiting sense of shame is a matter of daily observation. I feel sure, moreover, although I am told that the psychologist does not accept that view, that there is added to this more than a dash of normal primitive, atavistic, biologic cruelty and lack of sympathy, that in this connection takes the form of enjoying his mother's unhappiness. The Germans have a word for it, *Schaden Freude*, rejoicing in the discomfiture of others, an emotional reaction by no means wholly restricted to chil-

dren. It would be hard to conjure up a clearer picture of innocent cussedness and cruelty than that presented by one of these children resolutely refusing to open his mouth for food, vomiting purposely all over the table, or dawdling for hours over his breakfast, occasionally picking up a crumb while furtively watching his mother's distress. A little Jewish boy of about eight years of age came into my office not long ago and before his mother had a chance to begin the monotonous stereotyped tale of anorexia so familiar to the practicing pediatrician he said, "I don't eat. I can't eat what she cooks for me. I can eat if I go out somewhere else to eat." While he doubtless gloried in the distinction of being "a guy who don't eat," he must have known the cruel effect of such words and actions on his mother, who spent hours in trying to meet his peculiar reaction to her cooking. It is also improbable, again, that his body build, vitamin B intake, or the acidity of his stomach contents changed in walking from home to "somewhere else to eat."

The external irritant is the mother, or nurse, or father, trying to make the child eat what, when, and as much as each thinks he ought to. In innumerable homes there is a daily battle, often tridaily or oftener, with endless discussion over the dining table. On the one side the army advances with coaxing, teasing, urging, cajoling, spoofing, wheedling, begging, shaming, scolding, nagging, threatening, bribing, punishing, pointing out and demonstrating the excellence of the food, again weeping or pretending to weep, playing the victrola, singing a song, telling a story or showing a picture book, turning on the radio, beating a drum just as the food enters the mouth with the hope that it will keep going on instead of returning, even having grandmother dance a jig—all regularly recurrent actual procedures encountered daily. On the other side, a little tyrant resolutely holds the fort, either refusing to surrender or else capitulating only on his own terms. Two of his most powerful weapons of defense are vomiting and dawdling. Possibly there is back of it all in some instances a conditional reflex. When an infant, or an older child, is forced against his will to eat at a time when he is not hungry because of illness, fatigue, sleepiness, satiety, or what not, and it causes him to vomit either from necessity or as a matter of voluntary defense, as so often happens, it is not unreasonable to assume that vomiting and the fear of vomiting have become associated in his mind with the mere sight of food. I know of children who begin to gag, or pretend to gag, it is often impossible to tell which, as they approach the dining table. An extreme case will often serve as the best illustration. After examining a child in the home one day the usual question came up of what to do for the child because the school nurse had reported that the child was several pounds under weight. I told the mother not to

worry, not to make her eat, and said that the little girl would probably be as big and robust, some day, as her father, who was present. The father had a peculiar expression on his face, which led to the following story: Since a baby he had never been able to eat any fruit, vegetables, or meat. The sight of an apple especially causes him to shudder and a nearer approach nauseates him and gives him a feeling of terror. So strong was this reaction when he was a little boy that his brothers used to run after him with an apple, just as the bad boy terrifies the little girl by running after her with a snake. He drinks some milk and eats some cereal. His regular lunch consists of two or three dishes of ice cream and a Swiss cheese sandwich or two. This is commonly repeated at his evening meal. His wife took him in hand and tried to get him to eat vegetables, at least to drink some orange juice, but soon gave it up as a bad job and adjusted herself to an unchangeable habit complex. An invitation to dinner is obviously a near tragedy, with all the possibility of a real one if there should happen to be fruit on the table. In every other way he appeared to be a normal, healthy, prosperous, business man. I have not ventured to go into minor details with him. We have here an extreme illustration of the rôle psychologic factors in childhood play in moulding the adult along the lines under discussion. Innumerable variations and gradations all the way down to the normal are encountered daily on all sides.

The therapy of anorexia divides itself naturally into prophylaxis, and the curative treatment of the condition after it is established. Parenthetically, both of these procedures furnish the final proof that we are dealing with psychologic and not physical factors. The prophylaxis of anorexia can be expressed in one short pregnant sentence: *No child should ever be forced or coaxed to eat.* This applies to any age, except, of course, the first few days of life. There are exceptions in the case of severe illness. One occasionally encounters a child of whom it is stated that he never cared to eat from the day he was born—that he never had a normal reaction to the breast, or the bottle, or any other food. I have never been able to satisfy myself whether such children are *sui generis* or are the result of a faulty technic. I am inclined to the latter view. There are, unfortunately, for rather obvious reasons, few experimental or statistical data bearing on the prophylactic aspects of this subject. For this reason a report by Aldrich[8] is of peculiar interest. Of a consecutive series of 200 children whose mothers were told never to force or coax them to eat from birth on he reports that "only 15 per cent were reluctant eaters and none had developed an aversion to food. Not one in the entire group habitually or commonly vomited his food. And the average weight was 3.6 pounds above what is considered normal for

their age." This occurred in a good suburban community in which an estimate of 50 per cent of incidence of anorexia would probably err on the side of conservatism. It is too much to expect all mothers to follow scrupulously and at all times what might seem so radical a régime and I feel sure that a large part of the 15 per cent of "reluctant eaters" represented conscious or unconscious lapses on the part of mothers or nurses. It has been my own practice for many years to pursue this policy and to reiterate it on all occasions, and I am fully aware how hard it is to buck against a host of antagonistic forces.

The cure of anorexia, once firmly established, encounters many obstacles. We have not only the child to deal with, but what is far more important, his whole environment, of which his mother and (or) his nurse are the two important factors. The real difficulty and the real desideratum lie in changing that environment. The surest and most rapid cure, and there are few cures in medicine that are more sure and more rapid, is to send the child to a hospital ward with several other children of about the same age. The mother or former nurse must not even be allowed to visit the child. There is, however, a serious objection to this procedure. A hospital ward is not a wholly safe place to send a well child, and especially not a very young child. A summer camp, a foster home, sending him to relatives at a distance, letting him eat in the kitchen all suggest themselves. A long vacation, or other absence, on the part of parents, leaving the child alone with a strange nurse often leads to an immediate result. *In dealing with children there is a marvelous therapeutic virtue in strangeness.* That familiarity on the other hand breeds contempt has become a proverb. In the case of an infant one can commonly reach the desired goal by turning him over to a good, understanding, infant nurse in an otherwise unchanged environment except that the mother, or former caretaker, must appear only in the background and never at the child's meal times. Two methods of procedure lie open to the nurse in this situation, and it is usually best not to try to change the unchangeable in an infant nurse. It is usually easier to change nurses than to change a nurse. One is the method of feigned indifference, "lighting a Murad and appearing nonchalant." The other is to employ to the maximum the therapeutic value of strangeness and simply, but naturally and firmly, assuming that the child will eat because it is the thing to do. Either works if the nurse has the necessary gift for her particular method. I recently had an appointment to see a baby about a year of age. At the appointed time a man appeared at the door, looked furtively down the hall and then slipped into my office. He explained that he was the father and that he wanted to explain matters to me before seeing the child. It had become almost impossible to get the child to eat anything, and so he and his wife had gone else-

where to live, leaving the child alone with a nurse in their large apartment. He then stole out the back way and the nurse and child were admitted. The nurse was the good kind and said all was going well now that the parents were away. After the usual admonitions on my part she and the baby went out the front way. Soon the mother, who had parked in her car out of sight, came in to get my final verdict.

If no radical change in environment is feasible, as it commonly is not, then the difficulties are very real and, not infrequently, quite insurmountable. The first attempt, however, is usually made under such circumstances. One naturally sits down and has a long talk with the mother and (or) the nurse and gives them a brief synopsis of all that I have just said on the subject. Two simple methods of approach are available. Essentially they are the same. One is to appear utterly indifferent, apparently giving no thought whatever to whether he eats or not, or how much. After a given time, not too long, one proceeds nonchalantly and mechanically, but without question, to clear the table, whether he protests or not, but with the hope that he will protest. The other method is to give him for a time ridiculously small helpings only increasing them mechanically, again, as he will eat more, not because he demands more at first. Both methods aim to enlist his negative attitude in favor of eating instead of against it. The motive and all anxiety must, of course, be concealed. Both methods are equally applicable to the infant and the older child with such variations as are naturally imposed by the manner of taking food at a given age. There is nearly always a complete acquiescence and a promise to "try" it. One must then emphasize that "do" must be substituted for "try." Usually there is again complete acquiescence. It is, however, simply too much to expect an anxious mother to carry out a strange policy of not making her child eat when her whole concern is that he is already undernourished, is taking too little food as it is, and for a time will take even less while the hard-earned pounds that he just must gain according to the books, are slipping away. It is all the more hard if she is an intelligent modern mother who knows all about the importance of vitamins, and balanced diets, and the dire things that may happen if he does not get them. She may harden herself to a mechanical, nonchalant removal of food as soon as it is refused in the hope that it will stir up a negativistic demand for it, but it is hard to conceal the motive, and children are adepts at discovering motives. It is even harder to conceal her anxiety at all times, and this alone nullifies the whole procedure. That the father is not a great help in such domestic crises is a matter of common pediatric experience. No wonder that relapses are the rule on a "mother knows best" basis. That the results are not always unfavorable is proof of the pathogenesis I have indicated. Several years ago I saw a little girl

about three years of age with the usual complaint. I went all through the usual explanations with the mother and expected the usual result. I saw a little sister in the home a month later. The mother said nothing about the older child until I asked her about her eating. She said very simply, "Oh! She's fine—she's eating everything and has gained three pounds." Rather apprehensively, fearing she might have called in a Christian Science practitioner, I asked, "What did you do?" She seemed rather surprised at my question and answered, "Just what you told me to do." It was then my turn to be surprised. And yet I feel sure that this result could be obtained in nearly every instance if we had such cooperation.

Inseparably interwoven with all this is another psychologic factor. Man is by nature gregarious and especially is this true of children. The normal healthy exercise of that tendency is an association with those of nearly a like age. It is paradoxical that in a civilization that is becoming ever more crowded there is ever less constant, normal, free association with those of like age in early childhood. Two of the main factors are the small family and the inhibitions necessary to insure physical safety in childhood. The child is ever more thrown in contact with older people only. He cannot be trusted alone on the streets normally to seek his companions. The kindergarten and the preschool gatherings are after all a formal substitute for free normal childish activities. Eating with others, and especially with others of like age, promotes appetite; eating alone depresses it. A poor little rich girl of five years, whose constant, almost sole companion was a prim English governess of uncertain age, who was with her at breakfast, lunch and dinner, at the bath, at night, at the performance of that pathetic rite, when all dressed up she was led to her daily outing, actually gagged when she came near her solitary meals of well-chosen and well-prepared food. When the family went to Cape Cod and the child romped and played and ate with other children, she ate everything. When they came back she relapsed. One mother solved the problem of her child's not eating by employing a maid with a child of the same age as her own. Another mother made an arrangement with a mother in another apartment in the same building by which their two children ate together in one family one time and in the other the next time. Still another mother took her child to two different hotels or cafés each day. All of these instances were in one-child families, that pathetic exotic outgrowth of civilization with its almost inevitable hothouse environment that leads to a nearly 100 per cent of anorexia. Birth control is not without interest in this connection. The big old-fashioned family, with children normally spaced, rarely is troubled with anorexia.

The effect of herd psychology on anorexia and undernourishment was well shown in the days of the so-called "nutrition clinic." Those children did not eat together, but they did vie with one another in eating so that they might gain the necessary pounds that would add another coveted gold star at the showdown. The gold star in the home misses the point. It is like playing solitaire. The nutrition clinic did the job that lay before it and did it well. It gained the desired end by its appeal to mass psychology, and yet the nature of the appeal and the underlying principle on which it was based were as wrong as they were unnatural. It emphasized by extrinsic stimulation and exhortation a condition that is naturally and normally prevented and cured by the very omission of all such stimulation and exhortation.

There is a further dash of psychology that centers about the matter of choice and of surprise. Children love surprises. It is the essence of the Christmas present. We never outgrow it. To the housewife who does her own cooking the meal she has prepared does not have the same appeal that it does to her husband. To her it is a treat to go out to dinner and not know in advance what she is going to get. On the other hand, occasionally going to a café and choosing a meal is a stimulant to appetite. There is, however, something deadly about having to choose habitually from a stereotyped menu card three times a day. All of this applies with equal force to children, although the variations in reaction are wider and harder to predict. Especially important, it seems to me, is the matter of allowing children a larger freedom of choice and of variety in food. The younger child, especially if he has a pediatrician and an up-to-date mother, or nurse, is likely to get a monotonously definitely prescribed diet which the mother follows dutifully. I have never myself used diet lists for this reason. They are likely to mechanize a matter that normally and naturally calls for freedom of action based on general principles. There is, again, something deadly about a child's knowing in advance just about what he is going to get to eat at each meal, with only slight variations, and these often regularly recurring variations, such as fish on Friday, lamb chop on Tuesday, etc. Culinary ingenuity seems especially to have fallen flat in the matter of breakfasts. Mothers are prone to think that a child must have all of his vitamins, and all of the things that enter into a balanced diet daily and at just a certain hour of the day. This was pathetically illustrated in a recent appeal that was broadcast. We have been slow to realize, too, that babies and children of all ages have a wider tolerance for food than we have given them credit for. A realignment as to what is permissible and what is not in a child's diet seems to me clearly indicated. Our hospital dietitian once took a sort of poll of a number of wards as to what the children would most like to have to eat. The heaviest vote

was for "wienies and sauerkraut." They have them frequently as a result, and we have seen no harm from it. At another time she consulted me as to what foods I thought ought not to be given to children who were not acutely seriously ill. We finally decided to leave out pork roast and duck—but not pork chops. One must after all pay some heed to conventions. I have always allowed children in practice to eat their regular food if they cared to when sick, except, of course, in cases of appendicitis, or gastric ulcer, or similar conditions. I have always felt, too, that there was a distinct psychologic value in early convalescence when the appetite is not yet good in letting children have the things they want especially, even if they may seem a bit heavy, whatever that may be. There is always something amusing about including in a meticulous adherence to a liquid diet so uniquely solid a food as raw milk.

The following case has seemed to me of peculiar interest in this connection. We have at the present time in our wards a girl of eleven years with a chronic ulcerative colitis. Three months ago she was in desperate condition. I thought she was near the end. She had a few blood transfusions that apparently helped quite a little, some ultraviolet rays that may have helped some, and a fairly liberal diet. One day she overheard me say to the intern, "Let her have anything she wants to eat." The child interpreted this to mean that she could order anything she wanted in addition to her regular meals. She proceeded to do this and got it. One day she had three fruits for breakfast and three meats for dinner. She is very fond of corn. She eats what she wants to between meals. The change has been astounding. She gained two and one-quarter pounds the first week and twenty-three and one-half pounds in the first three months. She looks the picture of health. Her bowels are somewhat better—certainly no worse. We hope to build her up around her pathologic condition by treating her and not the local condition. She illustrates well what freedom of choice and competition with her own weight record will do in some instances.

Equally illuminating is the following: A young medical colleague was traveling in Europe with two children. One of these had been a serious problem in the matter of eating for such reasons as I have detailed above. He wrote me, "We were forced during a month of travel to let the boys eat largely what they pleased. They both gained weight and a victory. Now if they want nothing but steak for dinner they get it. . . . . The day after a meat orgy they asked for fruit and even—spinach! What problems we parents are!" Parenthetically, so much psychology has centered about spinach that one can readily agree with one of the four Marx brothers that "it would be better for the children of this country if the mothers ate the spinach."

I do not mean to infer that children can safely choose their own food by instinct, or under ordinary current domestic conditions, as we are told that the white rat does the proteins that contain the essential amino acids, or the hog the necessary components of an optimum diet if given a choice. That they can, however, safely choose their own food, under ideal conditions, if left unhampered in their choice of a large variety of foods that are unseasoned and contain among them all of the necessary nutritional ingredients has been convincingly demonstrated by Dr. Clara Davis[9] in an extensive experiment with which most of you are doubtless familiar. Fourteen children from six to eight months of age who had never had any other food than breast milk were allowed to select without restriction of any kind except that of three regular meal hours a day, their own diet for a period of years from a large assortment of cooked and raw, uncombined, unseasoned and unsophisticated foods consisting of such foodstuffs as one ordinarily associates only with the adult or older child. One child ate 10 eggs at one sitting. I saw one take 7 helpings of potato. Four or 5 bananas were a common occurrence. To me that experiment, extending over a period of five or six years, has been one of the most important contributions that has been made in the field of childhood dietetics and behavior in my time. It has reversed the usual process —instead of telling the child when, what, and how much he must eat, it has let him tell us what and how much a normal child will eat under normal conditions. Anorexia never occurred except with illness and disappeared with convalescence. No cathartic was ever necessary and there were no digestive disturbances. The problem did not exist. I saw them on a number of occasions, and they were the finest group of specimens, from a physical and behavior standpoint, that I have ever seen in children of that age.

While this procedure cannot in its entirety be employed in the home, the basic principle can nevertheless be utilized there to advantage. Dr. Davis has put this idea into practice in our orthopedic ward at the Children's Memorial Hospital, where conditions are more favorable, and it has been extended to some of the other wards. The food is wheeled into the middle of a large ward, à la cafeteria, and the children choose what they want and as often as they want to. It has led to a zest that this type of child does not commonly have. It has made of mealtime a sort of picnic, that has added materially to their enjoyment of life. They eat more. If they have three helpings of meat at one meal, they may eat six bananas at the next and so the nutritional budget will be balanced. This procedure recognizes another psychologic twist that is natural to all of us, I think, and that was very noticeable in Dr. Davis' experiment. We have waves of appetite of widely variable lengths in the presence of a free and wide

choice of food. I can best illustrate it if I may from my own experience. I went to Boston recently and had shad roe twice the same day. I have had none since. That was a short wave. For a number of years I used to lunch daily at a certain café. For several days I would take the same thing each noon, say a coffee crescent and coffee. This might be replaced for a few days by oyster stew, to be followed in turn, for a few days, by liederkranz cheese and rye bread. When we offer our children beyond the period of infancy a menu of the same daily pattern and content, it may be well to recall the story current in my childhood of the many men who had wagered that they could eat a quail a day for thirty days and had always lost the bet.

My theme has been a simple one. It is not the psychology of the books and the platform; it is the simple psychology of the home that lies an open book before us in daily practice. It can be seen fully only from within; it cannot be grasped at first hand from without. Therein the pediatrician has a point of vantage that the psychologist, the psychiatrist, the child guider, and the mental hygienist do not have in quite the same way. The pediatrician has not fully lived up to the obligation thus imposed upon him in this somewhat troubled period. I hope I have not added to the confusion.

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STOVARSOL IN THE PERORAL TREATMENT OF CONGENITAL SYPHILIS*

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HISTORY

AMONG the six hundred and five preparations with which Paul Ehrlich[1] worked before he arrived at the formula for salvarsan, "606," was a preparation known to him as number "594." In his experimental work Ehrlich considered number "594" unsuitable for intravenous usage, because it produced harmful results when injected under the skin of the mouse.

In 1921, E. Fourneau[2] published a preliminary report on the experimental use of this preparation in spirochetal and trypanosomal infections. This work was followed in 1922 by several publications by C. Levaditi and A. Navarro Martin,[3] L. Fournier, C. Levaditi, A. Navarro-Martin and A. Schwartz.[4]

These authors were able to show by experiments on animals, and later on man, that the *oral administration* of a preparation known to them as formula "190," chemically identical with Ehrlich's "594," was extremely well tolerated. They also demonstrated that when given per os, no such toxic and untoward reactions occurred as were found when it was administered intravenously. They were further able to demonstrate that the oral administration of this preparation had a definitely favorable therapeutic and prophylactic action on syphilitic infections in man.

The chemical formula for the preparation of "594" Ehrlich or "190" Levaditi, is acetylaminohydroxyphenylarsonic acid. The preparation is known under the general name of acetarsone, more specifically called stovarsol by the French, and spirocid by the German authors who have written on its usage. Stovarsol contains 27.1 to 27.4 per cent of arsenic.

There is not only a considerable literature on the use of this preparation as a prophylactic and therapeutic agent in syphilis, but there are also numerous reports of its usage in other spirochetal, trypanosomal and amebic infections. However, we wish here to consider only

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Merck and Company furnished the stovarsol used in this experiment.

the use of stovarsol in cases of congenital syphilis, and submit an experimental report of its administration in a series of cases which we have observed.

REVIEW OF THE LITERATURE ON USAGE OF ACETARSONE IN CONGENITAL SYPHILIS

There have been a number of reports in the French and German literature on the use of stovarsol in the prophylaxis and treatment of congenital syphilis. We have made a preliminary report,[5] but consider it essential to briefly review, in chronologic order, the reports of investigators in other countries, before presenting our own findings in detail.

Probably the first report on the use of stovarsol in the treatment of congenital syphilis was that made by H. Guillemot[6] in 1924, upon an eleven-month-old infant with condylomatous lesions about the anus and a fissure of the upper lip. A very striking improvement was noted six days after treatment was commenced, consisting of daily dosages by mouth of 0.02 to 0.07 gm. A total of 0.32 gm. of stovarsol was given orally to this infant in 13 days, with no untoward effects. Following this publication, Dupérié, Cadenaule and Clarac[7] reported the treatment of a two-month-old infant suffering with the following characteristic signs of congenital syphilis; snuffles, bloody nasal discharge, rhagades, condylomata, and pemphigus of the soles of the feet, together with identification of spirochetes from the serous discharges. A daily dosage of 0.12 gm. of stovarsol was given orally for twelve days, followed by a three-day rest period. Three such series were given, until the infant had received 4.5 grams, by mouth, in 66 days. At the end of this period of treatment all of the symptoms had disappeared and the infant had gained 1200 grams.

Krösl[8] reported his observations on 3 congenital syphilitic infants, and noted the complete disappearance of marked luetic symptoms by the peroral administration of stovarsol. He noted that the Wassermann reaction remained positive. P. Marfan[9] in his 1925 review on the treatment of congenital syphilis in infants noted the innovation of the oral administration of stovarsol. He recommended a dosage of 0.02 gm. per kilogram of body weight given in milk, one-half hour before a feeding or meal; the preparation to be given on four consecutive days of each week for a period of 6 to 8 weeks. He noted a mild diarrhea and an erythema of the buttocks in several cases, though the drug was generally well tolerated and easily taken by the infants.

Soldin and Lesser[10] (1925), reported in detail on a series of 7 congenital syphilitic infants and children treated with oral acetarsone (stovarsol). They noted the rapid disappearance of symptoms, the change in the Wassermann reaction of the blood serum and several untoward reactions in their cases. They started the dosage with 0.12 gm. per day and later increased to 0.25 gm. daily in infants under six months, to 0.5 gm. daily for babies over that age. They noted the disappearance of spirochetes in the serum of open lesions 48 hours after treatment had commenced. Two of the cases treated ended fatally; the authors attributing the deaths to intercurrent infections. They pointed out that the oral administration of stovarsol rapidly and favorably influenced the syphilitic symptoms, but had no effect on bronchopneumonia, otitis media, pyelitis or other concomitant infections. The same authors[10] (1928), in a second report were able to substantiate the earlier results they obtained with stovarsol by using a spirocid preparation. Both drugs have chemically identical formulas.

M. Oppenheim[11] was one of the earliest to report in the German literature, and has used this method in a large series of cases since 1926. In 1927, favorable reports on a series of congenital syphilitic infants and children were made by Klaften,[12] Steiner,[13] Scherber,[14] and Jaurros and Galarreta,[15] in 1928 by Krombach,[16] and in 1929 by Danzer,[17] Tuscherer[18] and Niederwieser.[19] In the most recent literature, from 1930 on, von Kiss,[20] Opitz,[21] Huber,[22] E. Müller,[23] Bratusch-Marrain,[24] A. Reuss,[25] Mettenheim,[26] Bamburger[27] and Nedelmann,[28] have submitted their results on the oral administration of stovarsol in the treatment of congenital syphilis.

DOSAGE

The dosage of stovarsol employed by these various authors has varied widely. There is a difference of opinion concerning the daily dosage of the drug, the total quantity to be administered, and the duration of the course of treatment. The daily dosage has varied from 0.01 gm. to 1 gm. daily; and the total amount given in the course of a cure, from 3 gm. to over 50 gm.

Some authors have recommended continuous daily dosages of stovarsol, administered for periods varying from six weeks to three months. Others have employed gradually increasing oral doses of the drug, giving courses of from seven to ten weeks' duration. These courses may be interrupted by rest periods of from four days to one week in length, during which time the administration of stovarsol is discontinued.

DOSAGE AND METHOD OF ADMINISTRATION

The arsenic content of stovarsol is approximately *27.1 to 27.4 per cent*, while the content of neoarsphenamine is 19 per cent and that of sulpharsphenamine is 21 per cent. From the above comparison, one can note the relatively high arsenic content of stovarsol.

The preparation which we employed was furnished to us by the manufacturers in the form of 0.1 gm. and 0.25 gm. tablets. Each 0.25 gm. (4 gr.) tablet contains approximately 0.068 gm. of arsenic, while the 0.1 gm. tablet contains approximately 0.027 gm. arsenic.

The dosage which we have followed in this series of cases approximates that suggested by Bratusch-Marrain[24] and is as follows: The first week the dosage is 0.005 gm. per kilo daily, or approximately $\frac{1}{13}$ gr. per kilo daily. The second week the dosage is increased to 0.010 gm. per kilo daily, or approximately $\frac{1}{7}$ gr. per kilo daily. The third week the dosage is increased to 0.015 gm. per kilo daily or approximately $\frac{1}{4}$ gr. per kilo, and in the fourth week the dosage is increased to 0.02 gm. per kilo daily, or approximately $\frac{1}{3}$ gr. per kilo, which is the maximum dosage used.

For the following five weeks the dosage is 0.02 gm. per kilo daily or approximately $\frac{1}{3}$ gr. per kilo daily.

The total dosage in our series for the nine weeks' course has varied from over 4 gm. to 42.5 gm., according to weight of child. This figured roughly to about 1 gm. per kilo of body weight for the whole course.

The individual course is of nine weeks' duration followed by a six weeks' rest period.

The method of administration has been as follows:

In the small infants, the mother was advised to crush the amount of stovarsol ordered, to dissolve it in water and to give it one-half hour before feeding.

When the dose ordered was large, in the older child, the instructions were that the medication be divided equally in 2 or 3 doses and be given with water, one-half hour before meals.

TABLE I

METHOD OF ORAL ADMINISTRATION OF STOVARSOL WITH DOSAGES

1ST PERIOD—7 DAYS	2ND PERIOD—7 DAYS	3RD PERIOD—7 DAYS
Stovarsol	*Stovarsol*	*Stovarsol*
0.005 gm. per kilo daily	0.010 gm. per kilo daily	0.015 gm. per kilo daily
1/13 grain per kilo daily	1/7 grain per kilo daily	1/4 grain per kilo daily

4TH PERIOD—7 DAYS	5TH PERIOD, 5TH, 6TH, 7TH, 8TH, AND 9TH WEEKS	6TH PERIOD
Stovarsol	*Stovarsol*	
0.02 gm. per kilo daily	0.02 gm. per kilo daily	Rest for 6 weeks
1/3 grain per kilo daily	1/3 grain per kilo daily	

Duration of entire active course	9 weeks
Duration of rest period	6 weeks

The reason for the administration of stovarsol before meals and with plenty of water is to prevent the too rapid absorption of the drug. It is to be noted that stovarsol in an acid medium, is rapidly decomposed and the free arsenic rapidly absorbed.

UNTOWARD EFFECTS

While there have been comparatively few untoward reactions experienced with oral administration of stovarsol, it is of utmost importance that they be recorded and borne in mind, so that their occurrence can be immediately recognized.

Mild reactions, such as slight elevations of temperature and diarrhea have been recorded. Klaften[12] and Krombach[16] were among the first to call attention to *Herxheimer reactions*, occasionally following the use of stovarsol. These disturbances clear up quickly when the medication is discontinued, and when the administration is again resumed do not tend to reappear.

Pezold[29] noted a rapidly disappearing exanthem after oral administration of stovarsol. Courtin[30] reports the case of a two-month-old congenital syphilitic infant who developed an arsenical dermatitis

with a severe bullous eruption. The medication was immediately discontinued, sodium thiosulphate was injected intravenously, but the infant became progressively weaker and died suddenly in a condition of collapse.

The same author reported another case in a 3½-month-old infant who developed on the tenth day of oral stovarsol administration, a fever, generalized arsenical dermatitis, a swelling of the subcutaneous tissues of the face, and infiltration of the skin of the entire body. By immediately discontinuing the oral administration of the stovarsol and by means of several intravenous injections of sodium thiosulphate, these symptoms of arsenic hypersensitivity were cleared up. From these reports it is apparent that hypersensitivity to arsenic as manifested by fever reactions, diarrhea and arsenical dermatitis may occasionally occur during the course of oral administration of stovarsol.

It is, therefore, important that patients undergoing arsenical treatments should be under close observation during the course of the medication. *It should be strongly emphasized that at the very first symptom of arsenic hypersensitivity, the medication be immediately stopped.* The course of treatment may, however, be again resumed after a sufficient interval of time has elapsed, after the cessation of all untoward symptoms.

COMMENT

The twenty-two cases in our series were seen at the pediatric dispensary of the Northwestern University Medical School.

All of the patients presented clinical findings of congenital syphilis and the diagnosis was corroborated by Wassermann and Kahn tests of the blood. Serologic tests were made routinely before the initial treatment was started, and thereafter, at the end of each six-week rest period following each course. Complete blood examinations were made before and after each course of treatment. Urinary examinations were made before the initial course of treatment was begun, and weekly examinations continued thereafter. X-ray pictures of the long bones were taken before treatment started, and at the end of each course. The weights and temperatures were noted at weekly intervals. All clinical manifestations such as snuffles, condylomata, skin eruptions, pseudoparalyses, bone changes, rhagades, fissures, keratitis, Hutchinson's teeth, dactylitis, splenic enlargement, were noted, and their time of improvement and disappearance during the course of the treatment was recorded. Untoward reactions were carefully watched for, and treatment immediately discontinued at the first sign of their appearance.

In our series, 14 patients were males and 8 were females. Fourteen were of colored race and 8 were of white race. The age variation was as follows: under 3 months, 4 cases; 6 months to 1 year, 3 cases; 1 to 2 years, 1 case; 2 to 3 years, 1 case; 3 to 6 years, 5 cases; and 6 to 12 years, 8 cases.

Of the symptoms noted in our series, 5 infants had snuffles, which disappeared from four to eight weeks after treatment was begun. Two infants had condylomata, which disappeared at fourteen and twenty-one days, respectively.

Six infants showed a generalized maculopapular syphilide, which disappeared from one to three weeks after treatment was started. Five infants had desquamation of the palms and soles, two of whom cleared up at the fourth week and three at the eighth week of treatment. One infant had had circinate lesions on the forehead for months, which disappeared after two weeks of treatment with stovarsol. Another infant showed punched out ulcerated lesions on the buttocks, thighs and legs, which were entirely healed two weeks after treatment began.

Four infants showed definite splenic enlargement. In three the spleen was no longer palpable at the end of six weeks, while one splenic enlargement persisted. Three children showed enlarged epitrochlear glands which could still be felt at the end of treatment.

Other clinical findings in our series were three children with mulberry molar teeth, one with Hutchinson's teeth, five with characteristic saddle nose. One child manifested frequent epileptiform eclamptic seizures, which became infrequent after treatment.

One must not overlook the fact that stovarsol has a marked beneficial effect on the physical development of the syphilitic infant and child. This has been noted by several authors, and prompted Niederweiser[18] and Tezner[31] to employ the drug in the treatment of nonsyphilitic dystrophic children, especially those prematurely born, with good results.

In our series of cases the average gain in weight for the nine weeks' course of treatment, was 4 pounds and 4 ounces. The greatest gain was 11 pounds in a nine-year-old child. One child five years old, gained 6½ pounds and one twelve-year-old child gained 7½ pounds. In the majority of the cases the general physical condition of the children improved markedly, they gained in weight, and their appetites improved.

In our series of twenty-two cases, the blood Wassermann and Kahn reactions before and after treatment were as follows: Wassermann before treatment was 4-plus in 16 cases; 3-plus in 5 cases; and nega-

tive in one case. The child with the negative Wassermann, who showed a 4-plus Kahn test, was a five-year-old child, whose mother is a general paretic with 4-plus Wassermann and Kahn tests and whose father has a 4-plus Wassermann and Kahn test. Both parents are deaf mutes.

The Kahn test before treatment was 4-plus in 17 cases, and 3-plus in five cases. In two it was not taken.

After one course of treatment the Wassermann reaction became negative in 13 cases out of the 22, and remained 4-plus in 9 cases. The Kahn test after one course of treatment became negative in six cases, 4-plus in 9; 3-plus in 3; and 2-plus in 2 cases. Most of our negative Wassermann and Kahn reactions after treatment were obtained in the infant group under one year. Of 6 infants under one year, five showed negative Wassermann and Kahn reactions after one course of treatment. One infant remained 4-plus to both tests.

Fourteen of our cases showed a negative urine analysis both before and after the course of treatment. Five cases had a negative urine before treatment, an occasional trace of albumin during treatment, and a negative urine after treatment. One case had a negative urine before and after treatment and showed a trace of albumin with occasional hyaline casts during the fifth week of treatment. One case showed from a trace to a 2-plus albumin before, during and after treatment. One case showed albumin, casts and red blood cells accompanying an arsenical dermatitis after the fourth week of treatment.

Routine blood examinations in our series revealed that the untreated young infants suffered from a marked secondary anemia. This is an agreement with the findings of other authors.[32] Following treatment the hemoglobin and erythrocyte values were found improved. In a ten-week-old twin the hemoglobin value which was between 50 and 60 per cent before treatment, rose to 60 to 70 per cent following the first course of peroral treatment.

Several of the children over two years of age showed initial anemia which improved following treatment. A twenty-seven-month-old baby showed a 50 per cent hemoglobin before treatment which rose to 70 per cent following treatment. A five-year-old child showed a rise of hemoglobin from 75 per cent before to 90 per cent after treatment and a twelve-year-old child showed a rise of from 75 per cent before to 85 per cent after treatment.

The majority of the older children showed little change in the blood findings. It was constantly noted that a rise in hemoglobin was accompanied by a proportionate increase in erythrocytes. The total leucocyte and differential counts were noteworthy only in so far as the eosinophiles were concerned.

Twelve children of this series showed an eosinophilia of from 1 to 9 per cent before treatment. The changes which occurred after treatment were as follows:

1. Of six children showing 1 per cent eosinophilia before treatment 1 showed 2 per cent after treatment.

2. Of three children showing 3 per cent eosinophilia before treatment 1 showed 4 per cent after treatment.

3. Of two children showing 4 per cent eosinophilia before treatment 1 showed 5 per cent after treatment.

Two children with no eosinophilia before treatment showed 3 per cent eosinophilia after treatment. The remainder of the series showed no eosinophilia after treatment.

SPINAL FLUID EXAMINATION

Fifteen of the twenty-two infants and children in this series had lumbar punctures performed after their course of treatment. The Wassermann and Kahn reactions on these fifteen spinal fluids were negative.

X-RAY FINDINGS

X-ray pictures were made of each patient in this series before treatment was begun, which included pictures of the long bones, the fingers of the hands, and where possible, also of the skull. After the course of treatment was concluded another series of films of the same bones were made. Of the twenty-two cases in our series, twelve were without osseous change in the x-ray films. Of these twelve, ten were children above four years of age, one infant was eighteen months of age, and one was two years old.

The remaining ten cases showed definite syphilitic changes as manifested by osseous lesions in the x-ray plates.[33] Six were infants under one year of age, one was twenty-seven months old and there were three children between four and six years of age. All of these patients showed periostitis, several showing the typical subperiosteal multilayered cloaking; in four infants a definite epiphysitis was noted as revealed by a widening of the provisional zone of calcification with submetaphyseal rarefaction. Several showed destructive lesions and foci of rarefaction at the metaphyses, while one infant showed a syphilitic dactylitis. One infant showed fractures of the ulna and tibia.

HEALING AFTER TREATMENT

Of the ten cases in our series, which showed osseous lesions by the x-ray, 4 were cases of Parrot's pseudoparalysis. These 4 cases of epiphysitis showed marked improvement clinically, after the first

week of treatment with stovarsol. The pain was less severe, swelling was less and motion of the affected part was greater. Clinically the symptoms disappeared in from one to three weeks.

Roentgenologically, the osseous changes were completely healed after one course of treatment. The infant showing a dactylitis improved greatly under treatment. In five weeks' time the swelling had disappeared. The bone changes as noted by the x-ray were completely healed after one course of treatment.

The cases with periostitis and those showing rarefaction and evidence of destructive changes in the bones, were completely healed after one course of treatment. All osseous lesions in our series were bilateral. We had no cases of tertiary bone lesions similar to those reported by Mettel.[34]

UNTOWARD REACTIONS

Untoward reactions were noted four times in this series. The first infant, a twin aged ten weeks, developed a slight diarrhea between the third and fourth week of treatment. Administration of stovarsol was discontinued for five days. The diarrhea promptly subsided, and no further reaction was noted. The second infant, also a twin aged ten weeks, developed a slight diarrhea between the seventh and eighth weeks of treatment. The medication was discontinued for three days and no other untoward symptoms developed. A third child aged five years, had concluded her first course of nine weeks' duration, when she developed a slight scaling and itching of the skin of the arms and chest. This mild skin reaction cleared up rapidly after application of a healing ointment.

The fourth reaction which occurred in a female colored child of six years of age proved most severe. The patient showed rhagades, periostitis and a 4-plus Wassermann and Kahn reaction before treatment. She weighed 46 pounds. For the first four weeks she took her treatment well, receiving a total of 6.5 gm. of stovarsol during this period. At this time she showed albumin, an occasional red blood cell and granular cast in the urine, and medication was immediately discontinued. A scaly rash developed over the chest and abdomen and treatment with intravenous injections of sodium thiosulphate solution was begun. Four days after development of the skin rash, diarrhea and abdominal pain were complained of. The temperature rose to 100° F. and the child looked toxic. The intravenous administration of sodium thiosulphate solution was continued, seven injections in all being given; the last sixteen days after the onset of untoward symptoms. At this time the patient looked much improved, the temperature was normal, she was no longer toxic, her skin was clearing, the diarrhea

and abdominal pain had ceased. On the seventeenth day after the onset of untoward symptoms the patient died suddenly. Autopsy was refused.

SUMMARY

Stovarsol (acetylaminohydroxyphenylarsonic acid) is a preparation containing 27.1 to 27.4 per cent of arsenic. We used this preparation, administered by mouth, in the treatment of congenital syphilis in a series of 22 infants and children.

The dosage of stovarsol in the treatment of congenital syphilis varies. In our series of 22 cases tested the following method was used:

The first week, 0.005 gm. per kilo of body weight was administered daily.

The second week, 0.010 gm. per kilo of body weight was administered daily.

The third week, 0.115 gm. per kilo of body weight was administered daily.

The fourth week, 0.020 gm. per kilo of body weight was administered daily (the maximum dose).

For the following five weeks the dosage was 0.02 gm. per kilo daily. The total duration of a course of treatment was nine weeks. A rest period of six weeks followed the treatment.

The method of administration is simple: In small infants the mother is advised to crush the amount of the drug ordered and to dissolve it in water and to give it one-half hour before feeding. In older children, the dose when larger, is divided equally in 2 or 3 doses, one-half hour before meals.

In our series of 22 cases, the clinical symptoms improved rapidly. Cutaneous lesions cleared in one to three weeks. The improvement in the general condition of the children was impressive. The appetite increased and the gain in weight was marked, average gain being 4 pounds 4 ounces for the nine weeks' course of treatment.

The Wassermann reaction in our series of cases after treatment with one course of stovarsol became negative in 59 per cent of the cases. Most of the negative Wassermann reactions were obtained in infants under one year. The Kahn test became negative in 30 per cent of the cases.

No serious urinary changes were noted in this series, with the exception of one child, who had a severe arsenical dermatitis. The blood of younger infants showed marked secondary anemia; improved rapidly with the treatment. Eosinophilia was a frequent finding in our series of cases.

The bone lesions healed rapidly after one course of treatment, both clinically and by x-ray. In cases of Parrot's pseudoparalysis the infant was able to move the affected extremity a few days after treatment was started and the swelling rapidly disappeared. There was complete healing after the end of treatment.

Two infants and one child showed mild untoward reactions which cleared rapidly with discontinuation of therapy. One child developed a severe arsenical dermatitis and appeared to be recovering when she died suddenly on the seventeenth day after onset.

SPINAL FLUID EXAMINATION

The Wassermann and Kahn reactions of the spinal fluid were negative after treatment on the 15 infants and children tested.

CONCLUSION

Stovarsol in the treatment of congenital syphilis is an effective and rapidly acting antisyphilitic remedy. The medication is easily administered and easily controlled. Our results warrant the belief that the peroral administration of stovarsol is the method of choice in the treatment of congenital syphilis.

In our clinic we have found the cooperation of our patients greatly increased by using this method. Previously, when congenital syphilitic children were treated by intramuscular therapy, we had great difficulty in having our patients return regularly for full courses of medication.

It should be strongly emphasized that patients undergoing treatment with stovarsol should be kept under close and careful observation. Parents should be warned that at the first sign of temperature, vomiting, diarrhea, or appearance of rash, the medication should be immediately discontinued. In this manner, any serious untoward effects can be eliminated.

The general physical condition of infants and children under this form of therapy improves rapidly, and the gain in weight is noteworthy.

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"PERNICIOUS" BLOOD PICTURES IN INFANCY

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DETROIT*

WE HAVE been interested for some years in a type of anemia occurring in infants from the third month to the end of the first year which presents a blood picture strikingly reminiscent of that seen in pernicious anemia. The cases have enough in common aside from the blood picture so that it seems worth while to present our series of eight as a group.

Our designation of the blood picture as "pernicious" implies, in the red cells, first, macrocytosis, common to all of the cases and marked in most. There is a good deal of variation in size, some microcytosis, and the kind of poikilocytosis usually associated with the pernicious anemia syndrome. Measurements from a typical case (Case 7) show: normal cells (about 7.5 μ) 48 per cent; macrocytes 42 per cent; microcytes 10 per cent. Second, high color index—always close to 1, and sometimes greater. The cells are well filled with hemoglobin and the smears show practically no achromia. Third, polychromatophilia, nearly always marked. Reticulocytosis sometimes runs to high figures. Fourth, normoblasts and megaloblasts. Nucleated forms were observed in all the cases. In one, an early one, the record does not distinguish types; in another the large nucleated forms are described as macroblasts. In the other six true megaloblasts in considerable numbers were seen.

The white cells varied more. Three patients showed marked leucopenia, while two had pronounced leucocytosis not accounted for by complicating infection. The differential counts were of little significance, though a few showed low percentages of polymorphs. The low platelet counts in the acute stage, without petechiae or hemorrhage, were striking. The details of the blood findings are given with the case histories.

Besides the blood picture, a second common feature to link these cases is the history. Six of the eight patients had histories definitely pointing to iron deficiency for one or another reason. The four who came under observation at less than six months of age were children of mothers who suffered during the pregnancy from anemia sufficiently severe so that it seems a fair assumption that the babe in utero could not lay down an adequate iron reserve. This assumption is supported

*From the Children's Hospital of Michigan. Presented at the forty-fourth annual meeting of the American Pediatric Society, May 26, 27, and 28, 1932.

in all these four by the absence of any other plausible reason for the anemia. Of the other four, more than six months old, there were two in whose cases iron deficiency was less obvious. One had a history of obstinate, untreated vomiting of spasmodic type, with inadequate feeding; and in the other (Case 6), who was offered an adequate diet, the trouble seems to have been the drain of prolonged infection, with lowered food intake. The last two had clear histories of lack of iron at the time when the reserve would naturally be exhausted, and one of them was, in addition, a prematurely born twin.

A third common feature in the six who survived was the surprisingly rapid, permanent recovery from seemingly grave anemia with no other treatment in any case than what might be supposed to be a temporizing procedure—transfusion, followed by appropriate diet. Iron was not given except in Case 3, where the patient, who had a prolonged ear infection some time after leaving the hospital, had it administered with the idea of forestalling recurrence of the anemia. Patient 7, the only one with prolonged hospital stay, was practically cured of the anemia in a short time. The long stay was due to a troublesome abscess.

A fourth feature was the rapid disappearance of abnormal red cells after one or two small transfusions.

The case histories follow, arranged in order of age rather than chronologically.

CASE 1.—A three-month-old boy of American parentage, admitted November 11, 1925, and discharged November 24, 1925.

(This, our first, was the private patient of another practitioner, and was treated in another hospital. The record is not very complete.)

Chief Complaints.—Pallor, vomiting and loose stools.

Family History.—The father was well. The mother, generally in good health, has been decidedly anemic during the pregnancy, for no more obvious reason than a capricious appetite. She stated that she had lived almost entirely on almonds. Her hemoglobin was about 60 per cent. There were two other living children, both well. One had died of pneumonia complicating Hirschsprung's disease.

Past History.—The birth was normal; birth weight 7 pounds 11 ounces. He was breast fed until admission, with some complemental feeding recently. He was always well nourished and fat. He breathed heavily (adenoid) and had received x-ray treatment for enlarged thymus.

Present Illness.—Pallor was noticed about three weeks before admission. Soon he began to vomit frequently and to have loose green stools. A blood examination showed 30 per cent hemoglobin, and the picture was said to be typical of pernicious anemia, with which diagnosis he was referred to us.

BLOOD

	HGB.	R.B.C.	W.B.C.	PMN.	EO.	LYM.	MACRO.	ANISO.	POIK.	MEG.	NORM.
11/11	4.2	1600000	14000	61	1	36	Many	++	++	+	+
	Platelets and reticulocytes not counted										
	Two transfusions, 75 c.c. and 80 c.c.										
11/20	10.6	3800000	12000	58	1	39	Few	+	+	+	0
4/5	12	4000000	12000	58	1	41	0	0	0	0	0

Physical Examination.—Weight 11 pounds 6 ounces. A well-nourished baby, with pronounced pallor. The only significant findings were nasal obstruction and palpable spleen. Urine and stools were negative.

The child was weaned during the hospital stay. The vomiting ceased promptly, and the loose stools soon after. He went out in good condition, and recent report states that he has never had further indications of anemia.

CASE 2.—A four-month-old girl of Scotch parentage, admitted December 14, 1925, and discharged December 17, 1925.

Family History.—The father was well. The mother was definitely anemic before and during the pregnancy. We have no information as to the cause or the blood findings. There were no other children.

Past History.—The birth was normal; birth weight 7 pounds. Feeding was one-half milk with Karo. Cod liver oil was begun two weeks before admission. There had been no illnesses. For one month before admission pallor and loss of weight had been noticed. Vomiting after each feeding had been present for two weeks.

Examination.—Weight 3930 grams. No important findings except undernutrition and pronounced pallor. Wassermann and Mantoux tests negative. Urine normal. Stools negative for blood and ova.

BLOOD

	HGB.	R.B.C.	W.B.C.	PMN.	EO.	LYM.	MACRO.	ANISO.	POIK.	RET.	POLYCH.	NUC. R.B.C.
12/14	4.2	1552000	4800	36	1	60	Many	++	+	30	++	3
	Transfusion 120 c.c.											
12/16	10.6	3400000	7700	20	8	71	Many	++	+	10	+	1
12/17	Discharged much improved.											
12/31	11.2	3400000	7200	25	2	73	Some	+	+	3	Little	0

This patient was sent in by a member of the staff for transfusion.

No other treatment was given. The vomiting ceased after the transfusion, and she gained 250 grams in the hospital. She is reported to have done well ever since.

CASE 3.—A five-month-old Scotch girl, admitted June 1, 1931, and discharged June 5, 1931.

Family History.—The mother had been anemic for fourteen years. Her hemoglobin June 2, 1931, was 27 per cent. She had albuminuria during this pregnancy. Her anemia yielded to treatment readily later. The father was healthy. There were two other children, both well.

Past History.—The birth was ordinary; birth weight 5 pounds 9 ounces. She had the breast two days, then S. M. A. until onset of vomiting; finally Pet milk with Karo. She had no acute illnesses; no diarrhea. About two months before admission pallor and loss of weight were observed; then vomiting of most of feedings, and green, undigested stools.

Examination.—Weight 4350 grams. Extreme, chalky pallor. Few pigmented spots on forehead, possibly remnants of purpura. Bilateral otitis media. All else negative. Wassermann and Mantoux tests negative. Stools negative for blood and ova. Urine normal.

During this admission the patient had a nasopharyngitis with slight fever. The ears were not opened. She was discharged in good condition. She was readmitted October 31, 1931, to be observed for possible mastoiditis. In the interim various counts had shown eosinophilia, going as high as 45 per cent, with no discoverable cause. She had a prolonged suppurative otitis, during which she was given iron. Her hemoglobin fell once to as low as 10 grams. The mastoids were not operated upon. At final discharge hemoglobin was 16 grams, the eosinophilia had disappeared, and the whole picture was normal. She had gained 3 kilograms.

BLOOD

	HGB.	R.B.C.	W.B.C.	PMN.	EO.	LYM.	MACRO.	ANISO.	POIK.	RET.	POLYCH.	PLATE.	MEG.	NORM.
6/1	4.2	890000	3750	37	0	57	Many	++	++	12	++	Few	1	5
6/1	Transfusion 75 c.c. 6/2 75 c.c.													
6/3	13.5	3830000	3950	17	9	67	Few	+	+	4	++	100000	1	4
6/3	Transfusion 55 c.c.													
6/5	14	4450000	5000	13	27	23	Few	+	+	4	+	50000	0	0

CASE 4.—A five-month-old boy of German extraction, admitted February 14, 1930, died February 18, 1930.

Family History.—The father was apparently well. The mother gave a history of anemia with splenomegaly since her sixteenth year. Examination proved this to be typical hemolytic icterus. She was fairly well during her first two pregnancies, and the children born of these were healthy. She was quite ill while pregnant with this patient, and on his admission had 8.5 grams hemoglobin. She has had treatment since, has gone through another pregnancy, and has a baby which seems perfectly well. None of the children have signs of hemolytic icterus. There is no history of tuberculosis, lues, etc.

Past History.—The birth was normal; birth weight 6 pounds 8 ounces. Feeding was breast plus dilute milk with Karo for one month, then bottle alone. He never took his feedings well, was fretful and cried a great deal. He was brought to the hospital with complaint of poor appetite, constipation and failure to gain.

Examination.—Weight 4 kilograms. An apathetic, malnourished baby. Very pale, with shiny, inelastic skin. Mouth-breather. Small palpable cervical glands. Signs of beginning rickets. Spleen palpable. Soft systolic murmur. Some râles at bases.

BLOOD

	HGB.	R.B.C.	W.B.C.	PMN.	EO.	LYM.	MACRO.	ANISO.	POIK.	RET.	POLYCH.	PLATE.	MEG.	NORM.
2/14	3.9	1240000	12600	9	3	73	Many	+	+	10	+	0	6	9
	Transfusion 100 c.c.													
2/17	10	3000000	13400	11	0	76	Few	+	+	10	+	0	6	4

The patient had no indications of hemolytic icterus. He developed pneumonia on the second hospital day, and died on the fourth. Complete autopsy was not allowed, but sections of spleen and bone marrow were obtained. The marrow showed very marked macrocytic and megaloblastic activity; the spleen had many areas of myeloid metaplasia.

CASE 5.—An American girl, seven months old, admitted July 19, 1926, and discharged July 26, 1926.

Family History.—Negative. The father, mother and four other children were in good health. There was no important disease history.

Past History.—The birth was normal—possibly overtime. Birth weight 9 pounds. Feeding: breast alone until recently; now milk thickened with cereal. Has had projectile vomiting since her third week; somewhat less on present feeding. Is fed with spoon. Has had no cod liver oil or orange juice. Could hold head up at one time, not now.

Present Illness.—Pallor was first noticed about three months before, at which time she had a slight cough. She was somewhat better until one week before, when generalized edema (probably nutritional) appeared, lasting several days. Her skin had been yellowish at times, but there had been no colorless stools or dark urine. No blood in excreta. Splenic tumor had been noticed recently.

Examination.—Weight 4960 grams. Undernourished; very pale. Slight icteric tinge in skin, not in sclerae. Small palpable cervical nodes. No rickets. Soft systolic murmur. The liver extends two fingers below rib margin; the spleen to crest of ilium. No other important findings. There was some pyuria on admission; no blood or bile. Icteric index 11; ind. van den Bergh test +.

BLOOD

	HGB.	R.B.C.	W.B.C.	PMN.	EO.	LYM.	MACRO.	ANISO.	POIK.	RET.	POLYCH.	PLATE.	MEG.	NORM.
7/19	2.1	1000000	23200	23	1	53	Many	++++	++	15	++++	0	20	2
	Transfusion 150 c.c.													
7/23	7.7	2400000	9200	20	1	71	Less	++	+	3.5	+	48000	6	2
	Transfusion 140 c.c.													
7/26	12.1	5300000	6100	19	2	76	Few	+	+	3	0	64000	2	1

She did not vomit during her hospital stay. The pyuria disappeared. She was discharged much improved, with good color. The spleen had receded to above the level of the navel. August 3, 1926, her blood showed 4000000 R.B.C., 12.6 hemoglobin, 11700 W.B.C., and no abnormal forms. The spleen was not palpable. On December 30, 1931, she was reported to have been in good health ever since.

CASE 6.—An Irish boy, ten months of age, admitted May 25, 1930, and discharged June 8, 1930.

Family History.—Wholly negative. No other children.

Past History.—Birth was normal; birth weight 8 pounds 4 ounces. Was breast fed one month, then bottle under the care of a pediatrician, with addition of cod liver oil, orange juice, cereals and vegetables at proper time. Apparently did very well until between six and seven months, when he had a large abscess on one thigh, followed by a succession of furuncles, the whole lasting somewhat more than six weeks. After this he was supposed to be doing well until the pallor became noticeable. He was sent to the hospital because of the anemia, which had been recognized for one month.

Examination.—Weight 9950 grams. Well-nourished child of normal behavior, but very pale. The only significant finding was that the spleen was readily palpable.

BLOOD

	HGB.	R.B.C.	W.B.C.	PMN.	EO.	LYM.	MACRO.	ANISO.	POIK.	RET.	POLYCH.	PLATE.	MEG.	NORM.
5/26	5.1	1600000	4100	34	1	65	Many	++++	+++	6	++	40000	3	1
	Transfusion 180 c.c.													
5/31	8.5	2600000	5000	50	4	44	Many	+	+	6	+	Few	0	1
6/3	10.2	3360000	4100	23	3	74	Few	Tr.	Tr.	2	0	75000	0	1

The patient developed a nasopharyngitis in the hospital, and was taken out against advice. He was seen recently, apparently in perfect health, and his blood was found to be normal. His improvement after leaving the hospital is said to have been rapid.

CASE 7.—A girl, ten months old, of German parentage, admitted October 28, 1931, and discharged December 20, 1931.

Family History.—The father, mother, and seven other children were living and well. There was no history of important diseases.

Past History.—The birth was normal; birth weight 8 pounds. She was breast fed two months, then given an evaporated milk mixture. She had never had any cod liver oil, orange juice, vegetables, or egg. She had had no definite illnesses, but

had been fretful and apathetic recently. She was brought to the hospital because of vomiting of three weeks' duration. The pallor had not been noticed.

Examination.—Weight 6960 grams. General nutrition fair; little fat. Somewhat dehydrated. Marked pallor. Nothing else significant except palpable liver and spleen.

BLOOD

	HGB.	R.B.C.	W.B.C.	PMN.	EO.	LYM.	MACRO.	ANISO.	POIK.	RET.	POLYCH.	PLATE.	MEG.	NORM.
10/28	5	1220000	12400	20	1	65	Many	+	+	5	++++	Few	2	2
	Transfusion 150 c.c.													
11/2	12.8	3250000	8700	41	4	51	Few	+	Tr.	2	+	50000	2	0
	Infection intervened here, and the cells reverted to previous state.													
11/10	10	3000000	21700	46	0	50	Many	++	+	5	+++	Few	5	0
11/17	Transfusion 160 c.c.													
11/20	14	3190000	8400	33	3	60	Few	+	+	2	+	200000	2	0
1/20	19	4500000	17400	44	8	48	0	0	0	0	0	250090	0	0

This child's blood was remarkable for very large macrocytes and unusually deep, brilliant polychromia.

The series of infections she had in the hospital comprised a severe stomatitis, daeryocystitis, and finally an extensive scalp abscess. Her food intake during this time was poor, but the vomiting, which had ceased after a few days, did not recur.

CASE 8.—An Irish boy, eleven months old, admitted March 30, 1932, died March 31.

Family History.—The father is healthy. The mother vomited throughout the pregnancy, but does not seem to have become especially anemic, and feels well now. The family is poor, and the babies have had very little proper food. There are no other children than the patient and his twin brother. The twin has an ordinary "nutritional" anemia. The history is negative for tuberculosis, lues, etc.

Past History.—The birth was premature; twin; birth weight 4 pounds. Prolonged resuscitation was necessary after birth. He was breast fed for two months, then given lactic acid milk. Cereals and vegetables were said to have been given after eight months, but questioning elicited that owing to poverty the supply was irregular and meager. He seemed to be doing well until about six weeks before, when he had some eruptive fever, followed by an obstinate cough. From that time he refused almost all food. About one month before he fell on his head. One week before he had a unilateral convulsion. There had been projectile vomiting for five days, on account of which he was brought to the hospital.

Examination.—He seemed acutely ill. There was no fever. He was dehydrated, and very pale. The fontanel bulged. Kernig and Brudzinski signs were positive. The neck was slightly stiff. The spleen was not felt. The spinal fluid was xanthochromic, with a moderate number of old red cells. The urine was negative. Diagnosis: cerebral hemorrhage; iron deficiency anemia.

BLOOD

HGB.	R.B.C.	W.B.C.	PMN.	EO.	LYM.	MACRO.	ANISO.	POIK.	RET.	POLYCH.	PLATE.	MACROBL.	NORM.
4.7	2500000	20000	69.5	0	18.5	Many	+++	+++	7	+	50000	5	7

This blood had considerable resemblance to that of erythroblastic anemia. There was striking poikilocytosis, a good deal of achromia, and quite marked unevenness in the distribution of hemoglobin. There were many normoblasts, no true megaloblasts, and a moderate number of macroblasts.

The child died nineteen hours after admission, apparently of cerebral hemorrhage. Complete autopsy was not allowed, but pieces of spleen and bone marrow were obtained. The marrow seemed atrophic. The spleen showed myeloid metaplasia.

These cases are the only ones with this picture of macrocytes, megaloblasts, and high color index that we have observed during a six-year period in a large clinic.

There has always been a question as to the occurrence of pernicious anemia in infancy and childhood. A few cases have been reported under that title, but so far as we know, there is not one of them in which modern diagnostic criteria have been rigidly applied, and we are inclined to accept the general dictum that true pernicious anemia

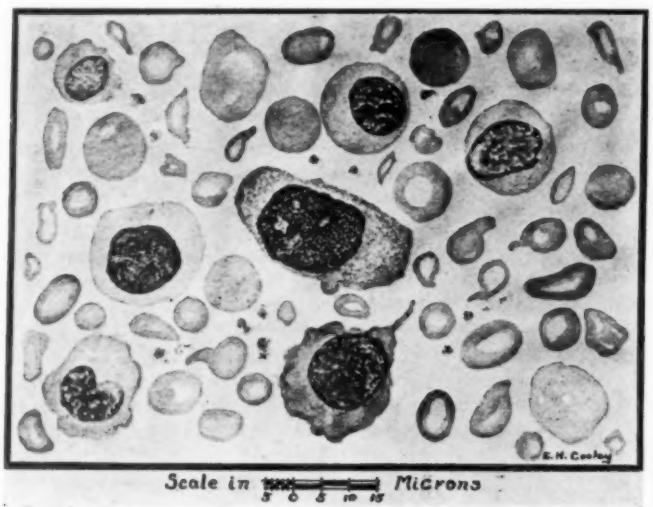

PLATE I.

Characteristic red cells from Case 6. The important features are macrocytosis, microcytosis, poikilocytosis, and megaloblasts in different stages of development.

is unknown in childhood. It is evident, however, that red cell findings such as we have described, especially when accompanied by leucopenia, as in Cases 2, 3, and 6, might well suggest this disease, and be quite misleading if death should supervene before opportunity for careful study had been given.

There is a considerable number of other case reports in the literature in which are described blood findings somewhat similar to ours—especially as to megaloblasts and normoblasts. The size of the cells is usually not mentioned. These cases do not appear under any one title. If, as in Case 5, there is splenomegaly and leucocytosis, they may be cited as examples of the von Jaksch syndrome. Otherwise they are

likely to be classed according to whatever feature is prominent in history or physical findings, as "syphilitic anemia" or "rachitic anemia," and the nucleated forms explained by the rather common assumption that the blood in any severe anemia of infancy tends to "revert to the fetal type." The histories are often vague on the points which have especially interested us, viz., the probability of an inadequate iron reserve, or of a failure to replenish that reserve at the proper period by furnishing iron in the food.

In regard to the question of insufficient iron reserve due to anemia in the mother; it is true that children of anemic mothers do not necessarily become anemic. Our first four cases, however, seem to present fairly good evidence that the mother's anemia may sometimes be an important factor, though it is not clear why this should be so rare.

From our experience we have come to doubt the correctness of the idea that, after the newborn period, the appearance of immature red cells in severe anemias is to be explained as reversion to fetal type. We are inclined to suggest that this particular blood picture is typical of a deficiency anemia—the deficiency in our cases being a lack of iron—and that the output of unripe cells is due to the inability of the marrow to produce enough matured cells with the material on hand. The argument for this view rests on the histories of the cases, the lack of other probable reason for the anemia, the remarkable effect of treatment by transfusion and diet, and the resemblance of the blood picture to that seen in other deficiency anemias. Plate I shows characteristic red cells from Case 6 of our series, while Plate II compares the cells of Case 7 with those of two other diseases characterized by macrocytes, megaloblasts and normoblasts. Pernicious anemia is now generally recognized as a deficiency disease; erythroblastic anemia we believe to be due to some congenital defect in absorption or metabolism; our view of the condition under discussion has been stated.

There is a much larger group of iron deficiency anemias occurring in infancy, to which most of the "nutritional anemias" of this period belong, and concerning which Mackay's valuable studies have given us some important information. They are usually of the "chlorotic" type, with low color index, are sometimes microcytic, and are commonly of slow development and moderate severity. We have seen no case with the macrocyte-megaloblast picture that seemed to belong here, unless Case 8 is considered to be of an intermediate type because of the somewhat different history and low color index. The importance of moderate variations in the color index is dubious, and we do not lay great stress upon them. We have no explanation for the differences between our cases and the commoner form unless it be the more rapid development and greater severity, which place a sudden stress upon the marrow.

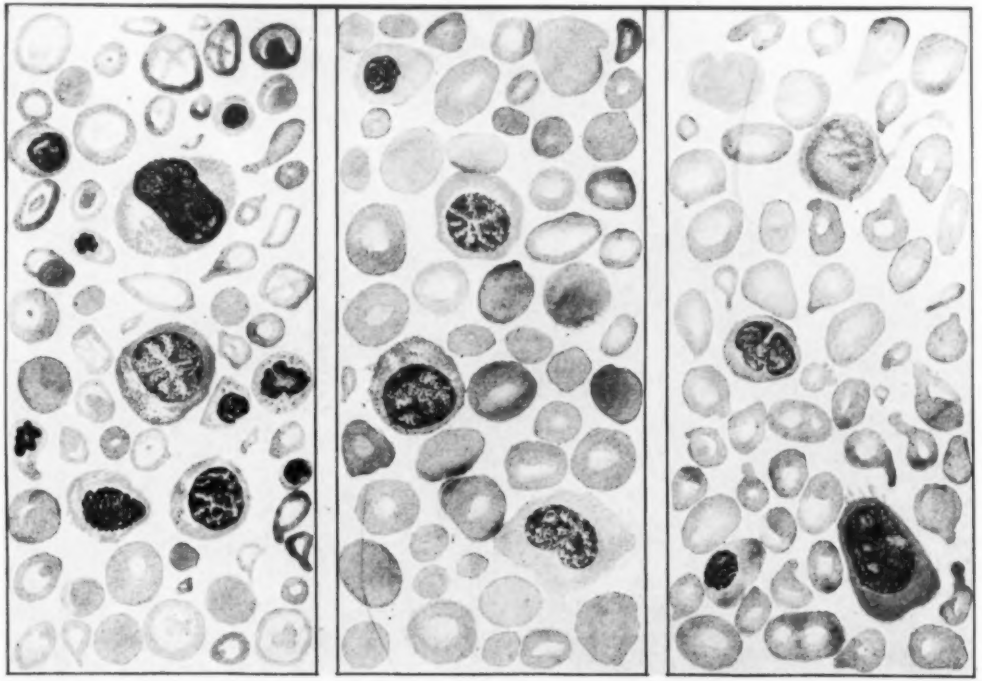

Fig. 1. Fig. 2. Fig. 3.

Scale Microns

PLATE II.

Plate II.

Comparison of cells from three types of macrocytic anemia.

Fig. 1.—From a case of erythroblastic anemia. Shows great variation in size, marked poikilocytosis, and the irregular achromia characteristic of the disease. Megaloblasts, macroblasts, and several forms of normoblasts are seen. Measurements: microcytes 12.8 per cent; normal, 50.1 per cent; macrocytes, 37.1 per cent.

Fig. 2.—From Case 7 (iron deficiency anemia). Macrocytosis is pronounced. Poikilocytosis is less than in Plate I. Several types of nucleated cells are shown. Measurements: microcytes, 10 per cent; normal, 48 per cent; macrocytes, 42 per cent.

Fig. 3.—From a case of pernicious anemia. Shows the macrocytosis and poikilocytosis of this disease, with megaloblasts and normoblasts. Measurements: microcytes, 8 per cent; normal, 70 per cent; macrocytes, 22 per cent.

Our method of treatment is based on what we believe to be the common observation that grave anemias are likely to be slow to respond to iron alone (or iron with copper, liver, etc.). We look on this as due to sluggishness of general metabolism when the oxygen carrier is inadequate. As we believe it desirable, to minimize infection and interference with growth, to cure the anemia as soon as possible, it is our practice to start treatment of our grave anemias with transfusion. Having found in the first two of these cases, which came within a month of each other, how easy it is to cure this particular condition by rather small transfusions and sensible diet, we have seen no reason to do anything more. We intend, however, to try iron alone, or iron, copper and liver, in appropriate cases. An interesting observation in this series has been that calculation of the actual gain in hemoglobin by means of blood volume estimations shows the increase to be almost exactly equal to the amount in the transfused blood.

The diet ordered for these babies is, of course, adapted to their ages. In general, it consists of whole milk or an appropriate mixture, with cereal, vegetables, egg yolk, meat once a day for the older ones, cod liver oil, orange juice, and some cooked fruits. Occasionally the milk was acidified with hydrochloric acid.

SUMMARY AND CONCLUSIONS

Eight cases are presented of anemia in infants in which the blood picture resembled that of pernicious anemia in the finding of macrocytes, megaloblasts, and (in all but one) high color index. Three showed leucopenia. The histories indicated iron deficiency as the cause of the anemia. Improvement after transfusion was rapid and abnormal cells disappeared promptly. The gain in hemoglobin was closely proportional to that contained in the transfused blood. There was no relapse when suitable diet was given. It is suggested that blood pictures of this kind are indicative of some deficiency which prevents the production of enough mature cells, and causes an output of unripe forms.

7815 E. JEFFERSON.

A SIMPLE, INEXPENSIVE STOCK FORMULA FOR YOUNG INFANTS*

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THE artificial feeding of young infants did not become a science until the beginning of the twentieth century when the Paris obstetrician, Pierre Budin, said: "In sterilized milk alone is safety and it must be the basis of all artificial feeding. The purest of milks, the sterile supply which flows from the mother's breast, given in excess, may cause fatal digestive troubles." His law, based on many years of clinical observation, states that there is danger in feeding more milk in twenty-four hours than the equivalent of one-tenth of the infant's body weight. For the first third of the century most of the artificially fed infants of France have been fed small, increasing quantities of undiluted, sterile milk.

Early in the century Heubner, Germany's first pediatrician, proved by means of the calorimeter, that the young infant requires approximately 100 calories per kilogram of body weight (about 45 calories per pound) in twenty-four hours. His one-third, one-half and two-thirds milk dilutions, enriched with lactose before sterilization, have retained their popularity for over a fourth of a century in many European countries. He stressed the danger of feeding more carbohydrate in twenty-four hours than the equivalent of one one-hundredth of the infant's body weight.

During the second decade, summer diarrhea with a high infant death rate from alimentary intoxication continued among artificially fed infants throughout Europe and America. Czerny and Keller put their faith in buttermilk, boiled with flour and sugar; Finkelstein and Meyer perfected Eiweiss-milch (protein milk); Soxhlet convinced the pediatric world that the combination of dextrin and maltose (Nährzucker) could be fed in larger amounts than could lactose, without provoking fermentation and diarrhea. Rotch's percentage method gained adherents in our Eastern States, but proved to be no panacea. By 1912 dextrimaltose had already replaced Nährzucker as the American carbohydrate of choice. A few years later Marriott found that the addition of lactic acid to sterilized cow's milk dilutions permitted the feeding of higher concentrations of milk and corn syrup. His formulas quickly gained in popularity and are today extensively used in the feeding of well and nutritionally disturbed infants.

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The most important advance during the third decade was Marriott's recognition of the virtues of unsweetened evaporated milk. His authoritative observations at once dispelled prejudice against such canned milk. Brennemann, a few years later, brought convincing evidence that excellent results could be obtained with evaporated milk, water and a vegetable carbohydrate, without the addition of lactic acid or any other acid. The gains in weight and the stools of many of his infants approached those of healthy breast-fed infants.

The present decade finds American journals rife with articles extolling evaporated milk dilutions. Favorable reports from all quarters have established for evaporated milk in a very brief time a singular place as a food for young infants. No adverse observations have thus far been published. All proposed formulas require periodic strengthening to suit age and weight increase. When the weight increase slows down or as an infant gets older it is customary for the physician to compute a stronger formula and the milk-room nurse or the mother makes the change. When the digestive apparatus has become adapted to the stronger mixture, weight increase proceeds.

The normal, breast-fed infant requires no such periodic food modifications; a slight increase in the amount consumed satiates him and his progress continues on the natural food without interruption. That this essential difference between formulas and breast milk has haunted European and American investigators is evinced by the numerous attempts from time to time to produce synthetic breast milk. The "perfect substitute" should resemble it not only in sterility, chemical composition and caloric value, but it should taste like breast milk, be readily prepared and inexpensive. When ingested, it should form small curds like those of breast milk; it should be quite readily digested, assimilated and tolerated. The stools should resemble in consistency, color, odor, and bacterial flora, those of the breast-fed infant. In brief, it should so closely approximate breast milk in all attributes that the only variable factor should be the gradual increase in the amount fed.

It seems paradoxical that no one has tried to enrich evaporated milk dilutions with lactose, the natural sugar occurring in breast milk. Nor has any one reported on a single dilution of evaporated milk, water and lactose, proportioned to approximate breast milk. It is about such a formula and the clinical results that this paper deals. In 1930, comparative tests were carried out to determine whether the rules of Budin, of Heubner, and of Soxhlet apply to excess of evaporated milk, of vegetable carbohydrate, and of lactose. Accurately computed, sterilized formulas were used. Within a few months it was found that excess of either component could elicit unfavorable intestinal symptoms. Infants, accurately controlled, showed individual differences in tolerance, but fretfulness, abdominal distention, flatus, slowing of the weight curve

and frequent, offensive stools could be induced at will in the majority of young infants. Excess of the evaporated milk—vegetable carbohydrate formula was not much better tolerated than was the same amount of milk enriched with an equivalent excess of lactose. Tolerance of our infants to excess of modern formulas is not unlike that of the infants of a previous generation—the admonitions of the old masters still apply.

The observations recorded below were made during the past eighteen months and embrace one hundred artificially fed private patients and fifty young institutional infants. The only formula used was one that so closely simulated breast milk that the results compare favorably with those observed in the nursling. In no instance did the food cause enteritis, nor did any infant fail to gain within a week after the formula was instituted. In recent months it has, to a great measure, replaced breast milk in prematurely born infants with an inadequate maternal supply.

TABLE I

THE BASIC FORMULA (STANDARD)

Evaporated milk	6 oz. (180 c.c.)
Water	12 oz. (360 c.c.)
Lactose*	1 oz. (30 g. or 2½ level, well-packed tablespoonfuls)

The initial amount of standard formula (Table III) is determined by the infant's net weight. During the first week (or less) the individual feedings are cautiously increased to the prescribed amount. If weight increase is not ample, if it lags, and if the gastrointestinal state of the individual warrants it, the formula is automatically increased to the next higher amount. The amount is never increased when the weekly gain is satisfactory.

For a group of infants the milk-room nurse checks daily each infant's feeding-card to determine the total number of ounces of formula required for twenty-four hours. This number is divided by eighteen for the number of ounces of lactose needed; six times the ounces of lactose equals the ounces of evaporated milk; twice the latter equals the ounces of water necessary for the entire twenty-four hours' supply. The ingredients are mixed by stirring until the lactose dissolves. The total number of ounces for each infant is measured off. It is divided into the required number of bottles. A strainer-funnel should be used. To sterilize the food before bottling it should boil in a deep, enameled saucepan for at lease five minutes, and sufficient sterile water should be added to replace water loss. If sterilized in the feeding bottles, only pyrex should be used. Satisfactory sterilization is possible by the aid of a sterilator, purchased at department stores. In the observations at the "Cradle" the feedings were autoclaved in 8-ounce

*If tolerance for carbohydrate is low (hot weather, enteritis), use two-thirds this amount of lactose.

pyrex nursing bottles at 15 pounds for 15 minutes. To insure absolute sterility of food and nipple, the latter is covered with a four-layer gauze square, secured by a rubber band (Fig. 1). Bacteriologic examination of unopened bottles at room temperature showed sterility at the end of one, two, and three weeks. Each infant's bottles are placed in tagged wire baskets before autoclaving, then cooled and refrigerated. It requires about an hour to prepare the twenty-four-hour

Fig. 1.—The feedings are autoclaved at 15 pounds for fifteen minutes. The gauze protecting the nipple is removed just before bottle is served.

supply for twelve infants. A quart of the standard formula costs about ten cents. At feeding time, the gauze is removed after the bottles have been warmed.

TABLE II

PERCENTAGE COMPOSITION (APPROXIMATE)

	STANDARD FORMULA	BREAST MILK
Fat	2.7	4.0
Lactose	8.7	7.0
Protein	2.3	1.5
Salts	0.3	0.2
Water	86.0	87.3
Total	100.0	100.0
Calories per ounce	20.5	21.0
Nutritive ratio	1:7	1:11

Observations on apparently normal infants of the same weight and age and fed equivalent amounts of the sterilized formula showed that they did not all gain at the same rate. The rate of gain is, to a great measure, dependent upon internal factors which may be termed "The innate power to gain weight and grow." It varies with the individual,

but is influenced by the quality, quantity and sterility of the food, the infant's health, and external factors as temperature, care, hygiene.

The longest continued gains in weight, without increasing the daily amount, did not occur with maximum amounts of formula per pound of body weight. Some infants gained consistently for months on 40 calories per pound of body weight; others required 50 calories; the majority made very satisfactory gains on 45 calories of the formula per

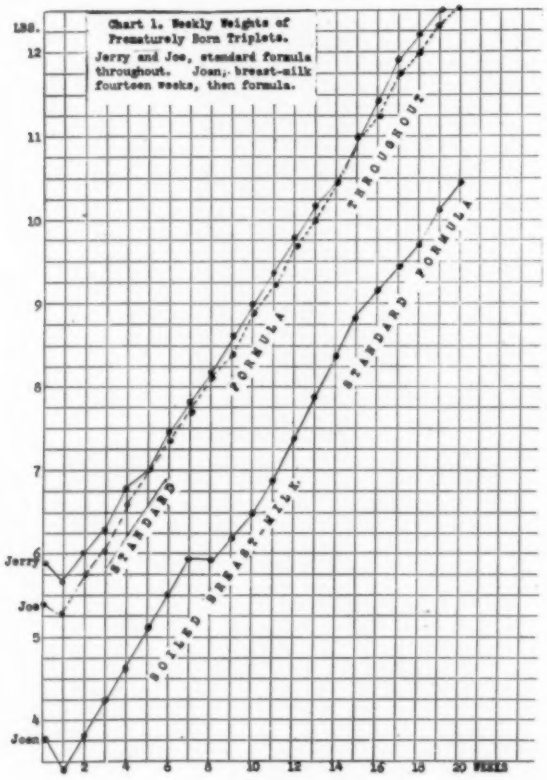

Chart 1.

pound of body weight in 24 hours. The majority responded to the standard formula as do nurslings. Too rapid increase, or premature increase (while an infant makes satisfactory progress) frequently deterred or inhibited weight increase (overstepping tolerance). If not rectified by prompt reduction of food the familiar picture of the overfed nursling could be duplicated (restlessness, gas, colic, abnormal stools). The infants in private practice averaged a gain of over a pound a month, many gained two pounds a month, some gained three

and more pounds a month. One gained six pounds in two months without an increase in the amount of food throughout that period of time.

Last year, prematurely born triplets (Chart 1) offered an exceptional opportunity to make observations with the standard formula. Two of the infants, Jerry and Joe, were fed the formula for twenty weeks. The sister, Joan, was fed boiled breast milk for fourteen weeks, then abruptly changed to the formula which was increased according to Table III as the weight increased. The weight chart illustrates how nearly identical the weekly gains of the boys continued. The girl's gain was not interrupted by the sudden change from breast milk to

TABLE III

TWENTY-FOUR-HOUR AMOUNT OF STANDARD FORMULA

INFANT'S WEIGHT IN POUNDS	TOTAL CALORIES @ 45 CAL. PER LB.	TOTAL OUNCES OF FORMULA @ 21 CAL. PER OZ.	FEEDINGS IN 24 HOURS
4	180	9	1⅓ oz. × 7
5	225	11	1½ oz. × 7
6	270	13	2 oz. × 6
7	315	16	2½ oz. × 6
8	360	18	3 oz. × 6
9	405	20	4 oz. × 5
10	450	22	4½ oz. × 5
11	495	24	5 oz. × 5
12	540	26	5½ oz. × 5

TABLE IV

TO COMPUTE THE PROPORTIONS FOR INFANTS OF VARIOUS WEIGHTS

TO MAKE	EVAPORATED MILK	TAKE OF	
		WATER	LACTOSE
12 oz.	4 oz.	8 oz.	1½ level packed tablespoonfuls
18 oz.	6 oz.	12 oz.	2½ level packed tablespoonfuls
24 oz.	8 oz.	16 oz.	3½ level packed tablespoonfuls
30 oz.	10 oz.	20 oz.	4½ level packed tablespoonfuls

After sterilization, replace water lost by evaporation.

standard formula. The stools of the three infants were usually so much alike in color, odor, and consistency that it was seldom possible to distinguish the breast-fed infant's. This unusual clinical test showed that the formula was practical if fed according to body weight.

The twenty-four-hour amount is estimated from the computed number of calories, based on the infant's net weight. The value of the food is about 21 calories per ounce; 45 calories per pound of body weight is the arbitrary initial amount (see Table III). The method permits flexibility. It is automatically increased to the next higher amount when the infant's weight increase lags. If the quantity nearest the net weight of the infant should not produce the desired weekly gain in weight and a food increase is indicated (increased age, absence

of anorexia, vomiting and frequent stools), the amount shown for the next higher weight is prescribed.

When the standard formula had given gratifying results in private practice, it was fed to young institutional infants. For a period of six months all young infants (without enteritis), entering Unit No. 3 of the Cradle,* were fed the standard formula. Each of the twelve infants in a unit is in an individual cubicle. Nurses and physicians wear mask and gown. Besides the established routine of weight, rectal temperature and stool records, additional individual notes were charted on the amount of food refused or vomited; the frequency, consistency, color and odor of the stools. When an infant failed to consume a feeding within twenty minutes, the bottle was taken away. During the first week the formula was gradually increased until the total for twenty-four hours equaled 45 calories per pound per day. Abdominal distention, regurgitation, flatus, colic and frequent stools, often encountered in the newborn during the first two months at the breast were conspicuous by their infrequency. When such symptoms were observed but two-thirds to one-half of each bottle was given as long as necessary; the prescribed amount was not resumed until improvement warranted. During the third week, 1 teaspoonful of fresh orange juice and 5 drops of Mead's viosterol (tested for sterility) were given daily in a little sterile water; during the fourth week, this was given twice daily; during the fifth week the original amount was usually trebled. The average admission age of the 50 infants was 9 days; the majority were less than one week old. The admission weights ranged from 4 to 10 pounds; the majority weighed between 6 and 8 pounds; the average admission weight was 7 pounds; 20 weighed less than 7

TABLE V

AVERAGE WEEKLY GAINS IN OUNCES
(50 "CRADLE" INFANTS ON STANDARD FORMULA FOR 300 FEEDING WEEKS)

INITIAL WEIGHTS	1	2	3	4	5	6 WEEKS
4 to 5 lbs.	−3 oz.	3 oz.	6 oz.	7 oz.	–	–
5 to 6 lbs.	3 oz.	5 oz.	6 oz.	5 oz.	7 oz.	6 oz.
6 to 7 lbs.	3 oz.	5 oz.	6 oz.	7 oz.	6 oz.	6 oz.
7 to 8 lbs.	3 oz.	7 oz.	7 oz.	7 oz.	6 oz.	7 oz.
8 to 9 lbs.	2 oz.	5 oz.	7 oz.	7 oz.	7 oz.	7 oz.
9 to 10 lbs.	3 oz.	4 oz.	5 oz.	6 oz.	5 oz.	6 oz.
Average	2 oz.	5 oz.	6 oz.	6 oz.	6 oz.	6 oz.

*The Cradle is an exceptionally well-equipped, regulated and personnelled placement-shelter, accommodating thirty-six newborn infants. To the management; to Miss Lopnow, the supervising nurse; to Mrs. Mulhern, in charge of Unit No. 3; and to Miss Meeker, in charge of the milk room, I wish to express my appreciation and thanks for their ceaseless cooperation and valued suggestions. The evaporated milk (Carnation and Pet brands) used throughout the observations at the "Cradle" was furnished gratis by the Evaporated Milk Association; the lactose was kindly donated by Merck and Company; the pyrex nursing bottles were given free by the Corning Glass Works.

pounds; 10 less than 6 pounds; 12 were prematurely born. Infants less than 6 pounds in weight were fed seven times; those from 6 to 8 pounds, usually six times; those above 8 or 9 pounds, five times in twenty-four hours.

All of the "Cradle" infants gained weight; none developed diarrhea. Table V shows the consistency of the average weekly gains. The smallest infants lost during the first week, and gained slowly during the second week. The slight gain recorded by each group during the first week is due to several factors. The initial feedings were cautiously increased to the prescribed amount in the course of several days or a week. Many of the newborns recorded weight loss during the first week (analogous to the so-called physiologic weight loss of nurslings), others refused to take the prescribed amount of food until they were a week or more old. Some infants, during the second and third weeks, showed a transient frequency of quite normal stools. It usually disappeared spontaneously within a week. In several instances the amount of food was slightly decreased for a few days. This condition has been noted frequently in nurslings during the first weeks of life.

DISCUSSION

Since Heubner's day it has been customary to start each artificially fed infant on some kind of a weak milk modification which is strengthened from time to time as tolerated and as the infant's weight increases. Indeed, the success of artificial feeding in recent years has been attributed in part to so-called "individualization"—the adaptation of the food to suit the infant. No one will deny the extreme importance of detailed study and the value of special formulas to combat specific disturbances of the gastrointestinal canal. Each infant with disturbed nutrition is a problem which deserves individual attention.

The observations of Budin show clearly that the normal, young, artificially fed infant can thrive on small, increasing quantities of undiluted, sterilized cow's milk.

The observations here reported indicate that the normal young infant can grow on artificial food without the customary strengthening of formulas. The substitute for breast milk recommended requires merely an increase in the daily quantity when the weight increase lags. The stools of infants fed exclusively on this formula from birth often resemble in appearance, color, consistency and odor those of breast-fed infants. Smears of such stools, stained by Gram's method, show a preponderance of gram-positive bacilli which resemble those found in the breast-fed infant's stool (B. bifidus or B. acidophilus). On many occasions the microscopic appearance of the stool was indistinguishable from breast stools.

CONCLUSIONS

A sterilized stock formula (basic formula: evaporated milk, 6 oz.; water, 12 oz.; lactose, 1 oz.) is quite like breast milk in composition, taste and digestibility. It has a value of approximately 21 calories per ounce. It is inexpensive. It is practical for the individual infant or for groups of young infants (maternity wards, day nurseries, and foundling homes). When the twenty-four-hour amount is kept between 40 and 50 calories per pound, the rate of gain is quite like that of nursing infants of the same age and weight.

636 CHURCH STREET.

NIRVANOL TREATMENT OF ACUTE CHOREA IN CHILDREN*

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IN PRIVATE practice and in the wards of the New York Post-Graduate Hospital and Medical School we have treated chorea with every reputable therapeutic measure, notably large doses of salicylates, sedatives (either bromide or luminal), and the various arsenical preparations. Wherever possible, cases were hospitalized and probable foci of infection removed; nevertheless the results were not satisfactory. Often the duration of the disease was not shortened, and serious cardiac complications were not prevented. However, with the introduction of the nirvanol therapy our results have been most gratifying.

Nirvanol, or phenylethylhydantoin (a combination of urea and glycol), was first recommended by Roeder[1] in 1919, for various nervous manifestations in children, such as wakefulness, irritability, and for reduction in the intensity and number of spasms in whooping cough, and the control of maladjusted movements in chorea. The best results were obtained in chorea vulgaris or Sydenham's chorea. Since 1919 there have been many reports of beneficial results following its use in this condition.

Leichentritt, Lengsfeld and Silverberg[2] of the Breslau Pediatric Clinic, reported the use of nirvanol in twenty children from four to thirteen years of age, with 80 per cent cures and 20 per cent greatly improved. They found nirvanol a valuable remedy, producing no ill effects on the cardiovascular system.

S. Schmal[3] reports good results in three very severe cases. Two of these had typical reactions, with improvement of symptoms; one case showed no reaction, but in ten days all choreic movements had ceased. Cardiac function remained normal and no urinary changes were noted.

Professor J. Gerstman,[4] Otto Beck,[5] J. Husler,[6] Kurt Matzdorff,[7] L. Keller,[8] Ashby,[9] and others all reported favorably on their results.

Poynton and Schlesinger[10] found beneficial effects in every case, especially those in the rheumatic group. Poynton found nirvanol more reliable than any other form of treatment. He believes it is more efficacious than antirheumatic drugs, antisera or vaccines, in that recov-

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ery is more rapid; cardiac lesions (if present) are favorably influenced; the disease is curtailed before the heart is involved; and recurrences may be prevented in a large number of cases.

For some reason or other it was not used in this country until the last year or two, and it was not until June, 1930, that Ray and Cunningham[11] published a report of its use in a series of cases. During the last two years we have used the drug in seventy-two cases, and in every case the choreiform movements ceased within twenty days after beginning the treatment. A larger number of cases could hardly be expected to show such universally good results, nor has time enough elapsed in these cases to show how many relapses there may be, although comparatively few have occurred to date. Leaving out of consideration the permanency of the results, we are convinced that it is the best treatment yet devised for controlling the individual attack.

It is our opinion that nirvanol is a specific for the individual attack of chorea. In all our cases the attacks terminated favorably and rapidly, complications were absent and the heart and renal functions remained undisturbed. The effects obtained from nirvanol therapy are obviously not hypnotic in character, but due to a specific action, presumably on the corpus striatum in the midbrain producing a definite reaction with changes in temperature, skin manifestations and characteristic blood changes.

The treatment is simple and available to all, since it consists only of the administration by mouth of a 5 grain tablet of nirvanol two or three times a day. The child is put to bed, kept on a simple diet, and if under six years of age, given a tablet night and morning, while larger children, over this age, are given three tablets a day, until a rash or fever or both are induced by the drug. This usually occurs on the seventh or eighth day, when the drug is stopped and no further treatment is given. A certain train of symptoms is produced by the drug, namely, drowsiness, fever, rash, leucopenia, eosinophilia, lymphocytosis. The drug is certainly unique since it is the only known drug that brings about a definite train of symptoms resembling one of the acute exanthemata.

Drowsiness begins on the third day when the child appears sleepy but rouses for food, urination, and defecation. Each day the drowsiness increases until in some instances the patient is almost stuporous and can be made to eat only with difficulty. In our first case we were alarmed by the intense stupor, but subsequent experience proved it to be harmless. Although the drowsiness varied in intensity, all of the patients could be roused to answer questions, or to take food. On the

seventh or eighth day when the drug is stopped, the drowsiness reaches its maximum point, gradually clearing up in the next four or five days when the patient again becomes alert and choreiform movements have disappeared. As the drowsiness begins, there is an exaggeration of the choreiform movements, that is, the chorea seems worse and a peculiar drawling speech develops. This speech peculiarity was seen in every case and resembles the speech of a severe epileptic such as occurs at times after a convulsion.

Fever usually starts on the seventh or eighth day and its advent is an indication for the withdrawal of the drug. In some of our cases the temperatures were as high as 104° F., in others 101° F. to 102° F., and there were a few cases that had no more than 100° F. fever at any time. The fever lasted four or five days after the drug was stopped and recurred in only one case. In this case it was probably due to some other cause. In thirteen of our cases the temperature was below 100° F., in fifteen cases between 100° F. and 101° F., and in thirty cases the temperature was over 102° F., and in one case it was as high as 106° F. Where the temperature ran very high, there was considerable prostration and usually a deeper stupor.

The rash began on the seventh to the ninth day, usually with scattered papules over the back and shoulders. These papules became macules which coalesced and gave an appearance much like measles, gradually spreading to the rest of the trunk and to the extremities. Later, or at some time during the febrile stage, a scarlatiniform rash appeared, usually on the face and sometimes covering the entire body. It was an intense, bright red rash closely simulating scarlet fever. The soft palate and the throat became injected and in some instances resembled the throat of scarlet fever. After four to six days, the rash gradually disappeared, leaving no exfoliation or other evidence of its severity. Nirvanol should always be discontinued at the onset of the rash even if unaccompanied by fever. Forty-nine of our cases had a maculopapular rash resembling measles or urticaria, while in seven cases there was a scarlatiniform rash only. The rash became hemorrhagic in three cases, but like measles it was not a serious complication and the petechiae disappeared in due course. Twelve of the cases had no rash whatever.

The blood findings during the reaction were characteristic. A leucopenia was present in all cases with a minimal white count of 2,000 and a minimal polymorphonuclear percentage of 20. The erythrocytes were not affected. The lymphocytes were increased in all cases, an average count being 40 per cent. Eosinophilia was the most constant finding during the reaction in those cases which did not show high

temperature or marked skin manifestations. The eosinophiles showed an average increase of from 4 per cent to 8 per cent, in one case increasing to 18 per cent. In fifteen cases the eosinophiles ran between 8 per cent and 12 per cent. Although the leucocyte count was often high, 12,000 to 15,000 before treatment, there was a considerable drop in the white count in every case. In one case the lowest white count was 13,000, but the average was between 2,000 and 7,000.

During the febrile stage, the percentage of CO_2 in the blood approached that of an acidosis in only one case—showing a drop from 46.5 to 27 per cent CO_2 in the blood plasma. The pulse did not vary appreciably. The urine examinations were negative in all cases. Headache and dizziness were observed in a few cases, and conjunctivitis and drowsiness were present in nearly all. Speech disturbances were improved with treatment. The cure is now complete for this attack. Recurrences, although infrequent, may appear at a later date and can be treated with nirvanol as during the initial attack.

Improvement may not be accompanied by the aforementioned reaction. The drug however should never be given longer than twelve days, even in cases in which no reaction is noted, because indefinite administration of it may produce injury to the bone marrow, and aleucemic blood changes. A secondary reaction similar to the primary one may occur between the seventeenth and thirty-first days, and especially in those cases exposed to the bright sun or artificial sunlight.

Of our seventy-two cases, forty-seven were females and twenty-five were males. Forty-six of the cases were between the ages of eight and twelve years, the youngest child treated being four years of age and the oldest fourteen.

Nearly half the cases had had one or more previous attacks, thirty-eight, or 52.8 per cent, being treated by us for the first attack. Nine cases had had one previous attack, one case seven previous attacks, and twenty-four cases, or one-third, from two to five previous attacks. Nearly half the cases (thirty-four) were admitted to the hospital during the summer, from May to September, while only nine cases were admitted during the months of December, January, and February.

The cases varied in severity but no patients with mild or fleeting choreiform movements were treated with nirvanol; that is, all of the cases were either moderately severe or of a severe type. Some of the children had to be placed in padded cribs to keep them from injuring themselves, and it was our observation that the most severe cases re-

covered as promptly and as thoroughly as the milder cases. In judging the severity of a case, we considered not only the intensity of the movements but the number of previous attacks and the duration of the attacks before they came to us for treatment. One child had had chorea off and on for six years and another for three years; that is, one attack following another so closely that it might be said that the child was never entirely free of the choreiform movements. Another child had shown choreiform movements almost continuously for eighteen months and still another for ten months. Our observation of these prolonged cases showed us that the treatment was just as effective and as prompt as in some of the less severe cases. In thirteen of the cases the duration of the attack before treatment was started was two weeks, while in thirty cases the duration was between one and four weeks. In five cases the attack had lasted for three months before our treatment was begun. The average duration of the disease before treatment was thirty days.

The etiologic factor of the chorea was definitely rheumatic in twenty-nine cases. In sixteen cases there was no known cause while in seven recurring attacks of tonsillitis had occurred. Other probable causes of the chorea were carious teeth, frequent recurring upper respiratory tract infections, and a few of the cases followed tonsillectomy. Six of the cases had had a definite history of shock or fright or sudden pain, and one had intestinal parasites.

DOSAGE

The dosage depended upon the age of the child, its size and weight, the severity of the attack, and, strangely enough, the weather conditions. We believe that many failures in the treatment or even reported unfavorable results, are due to improper dosage. We surmise that failure of the treatment reported by others is due to too small a dosage or to too short duration of the treatment, for we never gave less than 10 grains a day to children over six years of age, and all large, robust children over seven or eight years of age received 15 grains a day. The minimum dose given to any child was 25 grains and the maximum 215 grains. Many of the older children received 150 grains as a total dosage over a period of from seven to ten days. The average total dosage was 70 to 90 grains, 37 of the children, or about one half, receiving this dosage. Some of the children who were most prostrated or who became seemingly too lethargic or depressed, received the larger doses during the hot, humid summer days. After these experiences, we learned to give smaller doses during extreme, prostrating weather. The average length of treatment was six to ten days, always being governed by the onset of fever and rash unless

these symptoms failed to appear. One case was treated for twenty-one days because the rash and fever failed to appear, five cases were treated ten days and three cases received only four days' treatment.

Cardiac lesions were present before treatment in more than half of the cases (38) and none of these children were harmed by the nirvanol treatment. The 34 patients with normal hearts on admission did not develop any cardiac manifestations either during their stay in the hospital or while under observation in the follow-up clinic. In two cases sinus arrythmia was noted before treatment was started and seemed to improve with the administration of nirvanol, which we judged in these instances acted as a sedative.

Fatalities have been reported with this nirvanol treatment and we believe that they must have been due to (1) overdosage, (2) not stopping the drug soon enough, (3) giving the treatment during an acute complicating disturbance, (4) some cause other than nirvanol. One fatality that has been reported verbally to us occurred in a child suffering with a subacute bacterial endocarditis. Chorea being a manifestation of rheumatism, and rheumatism, through its cardiac complications, being the eventual cause of death in so many children, it is remarkable that no deaths have thus far occurred in any of our children. We would warn against giving the nirvanol treatment to children with decompensation or acute carditis in any form, and we were careful not to initiate the treatment during any febrile disturbance, keeping all children in bed under observation for at least two days before starting treatment.

Recurrences following nirvanol treatment so far have not been as frequent as with other forms of therapy. To date we have had thirteen recurrences (18 per cent), occurring six months to a year after treatment. In most of these cases the treatment was repeated with the same immediate subsidence of the choreiform movements. These recurrences are a distinct disappointment but the willingness with which the parents consented to a repetition of the treatment testifies to the relief achieved. It seems to us worth while to be able to promise a subsidence of the distressing symptoms.

SUMMARY

1. Seventy-two cases of chorea were treated with nirvanol.
2. The symptoms of all disappeared within twenty-one days.
3. No harmful effects of the treatment were observed in any case.
4. Thirteen recurrences have been observed up to date.

CONCLUSIONS

Our experiences with these seventy-two cases lead us to believe that nirvanol is the treatment of choice for the individual attack.

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NIRVANOL TREATMENT OF CHOREA*

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FEW diseases compete with chorea in the number of therapeutic measures that have been proposed. The dramatic nature of the disease has made it an attractive subject for research and study, and a voluminous literature has sprung from the earnest efforts of more than a century's study of various methods of treatments. The value of these numerous clinical procedures is difficult to ascertain, for as G. F. Still[1] says: "Chorea is one of the diseases in which recovery follows the use of so great a variety of drugs that one is tempted to wonder whether the child might not recover equally well with no drug at all."

A recent contribution to the treatment of chorea was made by Roeder,[2] who suggested the use of nirvanol in this condition. Her report was soon followed by many others[3, 4, 5] in which the value of the drug was highly extolled. Later studies[6-14] seem to confirm the earlier optimistic reports.

Since the nirvanol treatment of chorea has not had a sufficient trial to justify its approbation or condemnation, it is, therefore, desirable that unfavorable as well as favorable experiences with it be recorded. This report deals with our experience with this drug in six cases in which there was not a single instance of improvement following its use.

Early in 1931 we adopted the plan of treating with nirvanol every case of chorea admitted to our ward. So that they might be more closely observed, no more than two cases were accepted for simultaneous study. During the control period and the treatment period which followed, independent observations were made by the authors as well as by Dr. Henry G. Poncher and the nurse in charge of the ward. There was a unanimous agreement of clinical impressions throughout the course of this experiment.

Six children, aged seven, eight, eight, nine, nine, and twelve years respectively, with acute manifestations of chorea were treated. The attack in two of these children was an acute exacerbation of a chronic type of the disease. One case was so severe that it was necessary to guard the sides of the bed to prevent the patient from throwing herself out upon the floor. We have designated her as a ++++ severity. Four other cases were unable to feed themselves, and they have been designated as +++. One case was less severe, and we have classed her as ++. (See Table I.)

*From the Department of Pediatrics, University of Illinois College of Medicine. Submitted for publication, June 6, 1932.

TABLE I

DATA IN REGARD TO NIRVANOL THERAPY AND SICKNESS

CASE	SEVERITY OF CHOREA	DURATION OF CHOREA	NIRVANOL DOSAGE			NIRVANOL EFFECTS				
			NO. DAYS	DAILY DOSE	TOTAL DOSE	RASH	TEMPERATURE	STUPOR	LEUCOPENIA	EOSINOPHILIA
1	+++	7 weeks	21	0.3 × 8 days 0.45 × 13 days	8.25	+	+	+	0	0
2	++	4 weeks	8	0.3 × 8 days	2.5	local ++++	100.8° ++++	0	++++	++++
3	+++	3 weeks	11	0.3 × 7 days 0.45 × 4 days	3.9	++++	103.0° +++	0	+++	8.0% ++++
4	+++	?	18	0.3 × 13 days 0.9 × 3 days 0.6 × 2 days	7.8	0	102.8° ++ furuncles	++++	++	9.5% +++ 5.5%
5	++++	7 weeks	21	0.3 × 8 days 0.45 × 5 days 0.6 × 8 days	9.4	+ local	101.4° 0	+	0	+++ 7.5%
6	+++	1 month	8	0.3 × 8 days	2.4	++++	++++ 104.0°	++	0	++++ 9.5%

CONTROL PERIOD

These children were admitted to the hospital and were observed for a period of time varying from four to thirteen days. The duration of the control period was six days for two cases, four days for two cases, five days for one case, and thirteen days for another. During this time no medication whatsoever was given. This control period was employed to determine the effect of simple bed rest, and to obtain a temperature curve and blood counts in the absence of active therapy. In no case was there any improvement noted during this period of observation. An elevation of temperature was noted in only one case (M. H.), and this was due to furunculosis. At least three complete blood counts were taken during the control period. There was an elevated white count in only two patients. (Case 2 and 5), and they had an average count during this period of 8,600 and 11,200 respectively. The eosinophiles were uniformly low. The highest average eosinophile count obtained during this period was 3.5 per cent, and two patients had an average count of 0.6 per cent. (See Table II.)

TABLE II

EFFECT OF NIRVANOL ON THE BLOOD COUNT

C—CONTROL PERIOD N—NIRVANOL PERIOD		WHITE BLOOD COUNT			EOSINOPHILES			NO. OF COUNTS
		MAXIMUM	MINIMUM	AVERAGE	MAXIMUM	MINIMUM	AVERAGE	
Case 1	C	7600	6600	7080	1.5	0	0.6	3
	N	9400	6000		3.5	0		15
Case 2	C	12800	6500	8600	2.0	1.0	1.4	5
	N	9400	3200		8.0	1.0		16
Case 3	C	8100	7000	7400	4.0	3.0	3.5	5
	N	8300	4300		9.5	2.0		10
Case 4	C	7300	6500	6900	1.0	0	0.6	4
	N	11100	4700		5.5	0.5		15
Case 5	C	13100	10500	11280	3.5	2.0	2.7	4
	N	12900	7500		7.5	0.3		14
Case 6	C	7700	4500	5720	1.5	1.0	1.6	4
	N	7900	4000		9.5	2.0		13

THE NIRVANOL PERIOD

The powdered drug was routinely administered in dosage of 0.1 gm. three times a day for at least seven or eight days, and if a nirvanol effect was obtained in that time, the treatment was discontinued. If, however, the effect was not obtained, the dose was increased and continued for a maximum of twenty-one days.

It has been recommended that the drug be given until nirvanol sickness develops. The accepted signs of the disease are rash, temperature, stupor, leucopenia, and eosinophilia.

RASH

The rash is not unlike that which occurs in measles and in some cases is associated with a mucous membrane inflammation. Only three of the six cases developed a marked and definite rash, which appeared after six to ten days and lasted from three to six days. These three cases developed a rather high temperature. In two other cases the rash was only local. One case (Case 2) had mucous membrane lesions and desquamation and petechiae. (See Table I.)

CENTRAL NERVOUS SYSTEM EFFECTS

Central nervous system effects were obtained in only four of the children, and in only one case (Case 4) was there anything approaching a stupor; this appeared on the eighteenth day after 7.8 gm. of the drug were given and continued for two days. Marked drowsiness of four days' duration was obtained in one case (Case 6) after 2.4 gm. of the drug were administered over a period of eight days. Two cases had a very mild drowsiness; this appeared on the third day and lasted for eighteen days in one case, and in the other case it appeared on the thirteenth day and was present for only one day. (See Table I.)

BLOOD COUNTS

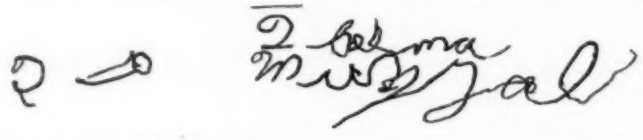
During the course of this experiment the six cases had a total number of 108 complete blood counts, 73 of which were made during the period of active treatment. The minimum number of complete blood counts made for any case was fifteen. An increase in total leucocytes during the treatment period was noted in only one case (Case 4). The maximum white blood count in two cases (Cases 4 and 5) was greater during the treatment period than it was during the control period. In general, however, there was a tendency for reduction of leucocytes. In every instance the lowest white count during the period of treatment was lower than it was during the control period, and the amount of the reduction seemed to be proportional to the intensity of the other symptoms of nirvanol sickness as is shown in Table I. The lowest white count obtained was 3200 in Case 2. (See Table II.)

SEQUENCE OF SYMPTOMS

There was no uniformity in the order in which the various symptoms manifested themselves, nor was there a complete correlation of the intensity of the various manifestations. The three cases with a severe rash had the highest temperature reactions and had the greatest degree of eosinophilia. The case with the most intense stupor had a fairly marked eosinophilia and leucopenia but had no rash.

OTHER THERAPY

Typhoid vaccine was given intravenously to two patients after they failed to respond to nirvanol therapy. In one case, a period of thirty-three days elapsed between the end of the nirvanol period and the beginning of the foreign protein therapy. Handwriting specimens of this patient are to be seen in Fig. 1. In the second case, there was an interval of eleven days between the nirvanol period and the foreign protein period. In both cases improvement was spectacular and im-

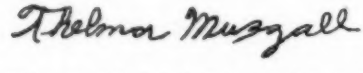


After Nirvanol Temperature Reaction

At Termination Of Nirvanol Treatment

After First Vaccine Treatment

After Second Vaccine Treatment



After Third Vaccine Treatment

Fig. 1.—Handwriting specimens of Case 3.

mediate following the vaccine therapy. Another patient (Case 6) included in this series returned to the hospital with a second acute exacerbation of chorea. At this visit she was given a course of foreign protein therapy to which she failed to respond. She was then given another course of nirvanol treatment and this time received 3.15 gm. of the drug in seven days. On this high dose, she failed to develop any signs of nirvanol sickness except a mild eosinophilia and a local rash. During the first course of treatment she developed all of these signs on only 2.4 gm. of the drug. There was also a failure of improvement after the second course of treatment.

SUMMARY

Six children varying in age from seven to twelve years were treated with nirvanol. In every case some sign of nirvanol sickness was obtained. Total dosage varied from 2.4 gm. to 9.4 gm. Three patients who did not promptly develop the signs were then given larger doses. In no case was improvement noted after administration of the drug.

Three children were treated after an interval of time with intravenous injections of dead typhoid bacilli. In two of these cases prompt and spectacular improvement occurred.

CONCLUSION

In our experience the treatment of chorea with nirvanol has been consistently without favorable results even though the drug was given in the accepted therapeutic amounts and definite signs of nirvanol effect were obtained. Certain cases responded satisfactorily to foreign protein therapy after nirvanol therapy had failed.

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THE ADDIS SEDIMENT COUNT IN CHILDREN

A Means of Determining the Presence of a Kidney Lesion*†

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INTRODUCTION

THE finding of albumin and cellular elements in the urine of infants and children with acute infections is a common experience. The term "febrile albuminuria" is used more frequently in the practice of pediatries than in adult medicine and is used in the former with little or no unfavorable prognostic significance. Particularly does severe dehydration, so much more frequently encountered in infants and children than in adults, lead to an apparent increase in urinary albumin and sediment.

Schloss[1] has reported an excellent study of the impaired renal function during intestinal intoxication in infants. There one will for the purposes of this study find adequate reference to the histologic findings in the kidneys of such infants. In summarizing his findings, Schloss concluded that the renal lesions in intestinal intoxication are not sufficient to account for the impaired kidney function. He specifically calls attention to the apparent greater increase in casts than might be expected from the degree of albuminuria but adds that the scanty urine might have been a factor in making the increase appear greater than it actually was.

In 1923, Wilcox and Lyttle[2] reported a study of kidney function in infants and children during acute infections. This study was based largely on the retention of nitrogenous waste products. The retention observed in about half their cases was attributed to simple impairment or depression of kidney function rather than to the presence of an anatomic lesion.

With our better understanding of the effect of urine volume and protein metabolism on the retention of nitrogenous products eliminated by the kidney, resulting from the work of Addis and collaborators,[3, 4] Van Slyke and collaborators,[5, 6] Rehberg,[7, 8] Adolph[9] and others, there seems little doubt that the decrease in urine volume and increased nitrogen catabolism during such acute infections and states of dehydration could explain the greater part of the retentions observed.

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We are not, however, so sure of our position when we discuss the urinary sediment in such cases, either from the standpoint of the actual change in the urinary sediment or the relation between any observed increase and structural changes in the kidney.

In 1925, Addis[10] introduced a method of quantitatively describing urinary sediment excretion and established normal standards for the adult individual.[11] He also showed[10] that the examination of the urinary sediment in patients with Bright's disease enabled one to determine the type of renal lesion during the course of the disease. As a result of these studies Addis[12] defined Bright's disease on the basis of a quantitative, not qualitative, difference between the urine of patients with Bright's disease and the urine of normal individuals. "Those individuals have Bright's disease who have more protein and a greater number of casts than are to be found in the urine during health." Specifically he places the excretion of more than 30 mg. of protein and five thousand casts per twelve hours as above the normal range.

To our knowledge no quantitative urine sediment counts have been reported for infants and children, either for the determination of normal standards or in patients where it would be interesting to know whether the apparent increase in the urine sediment, so commonly observed, was actually present when determined by quantitative methods. Data on this latter point would be added information in regard to the applicability to children of Addis' definition of the presence of Bright's disease in an adult, and would of course be equally helpful in judging the correctness of Schloss' opinion, when in discussing the evidence for kidney disease in intestinal intoxication,[1] he concluded that "if definite anatomic lesions are the sole criteria of kidney disease, we should not be warranted in assuming that a kidney lesion was present solely on the basis of albuminuria and cylindruria."

The present study has been undertaken for the purpose of obtaining such quantitative urine sediment counts from children as would seem to be of interest from the above considerations. The study falls into the consideration of four groups of patients; one, the normal child; two, the child with acute infection; three, the dehydrated child; and four, the child with Bright's disease.

In this paper we have presented data obtained on children falling within groups one and two together with a few counts on patients with Bright's disease. Data on patients in groups three and four will be presented in a later paper. We have also presented in this paper a slight modification of Addis' technic which we feel contributes to the accuracy of the counts in the particular respect in which greater accuracy is desired, namely the differentiation of red and white cells.

EXPERIMENTAL PROCEDURE

The method of obtaining the urine specimens and performing the sediment counts was based upon Addis' technic.[10] As we were dealing with children whose water metabolism is more labile than that of adults, we altered the experimental conditions slightly, by reducing the duration of the dehydrating fore period. The actual conditions under which the specimens were obtained unless otherwise specified were as follows:

The patient was allowed his usual lunch at 11:30 A.M. Following this, no fluids (defined as anything that pours) were given until 8 A.M. the following morning. A dry supper was given at 5 P.M. An attempt was made to avoid emptying the bladder from 2 P.M. until 8 P.M. The bladder was emptied at 8 P.M. and the urine discarded. An attempt was again made to avoid emptying the bladder until 8 A.M. All the urine voided between 8 P.M. and 8 A.M. was saved. If urine was voided between these hours, it was immediately put in a specimen bottle, containing a few cubic centimeters of toluol, and placed in the ice box. The specimens so obtained and preserved were, if acid, used for the counts. If the specimens so obtained were not acid, the collection was repeated under conditions favorable to the production of an acid urine.

In performing the actual count we encountered frequent disagreement between observers in the differentiation of red blood cells and white blood cells. There is usually a certain degree of uncertainty in the differentiation of red blood cells and lymphocytes in the ordinary examination of urine sediments, and, where the desired degree of accuracy is increased, as in quantitative counts, this uncertainty becomes of importance. We, therefore, made use of brilliant cresyl blue for the differential staining of the cells. The actual count was performed as follows:

The volume of the twelve-hour specimen was first measured to the nearest cubic centimeter. It was then thoroughly mixed and exactly 10 c.c. of urine placed in an accurately graduated 15 c.c. centrifuge tube, the graduations reading to 0.1 c.c. This sample was centrifuged at 1800 revolutions per minute for ten minutes. The supernatant urine was then carefully drawn off by suction until exactly 0.5 c.c. of urine was left in the centrifuge tube. Four and one-half c.c. of sodium citrate sodium chloride reagent* and four drops of freshly filtered 1 per cent brilliant cresyl blue in physiologic saline were added. The contents of the centrifuge tube were then mixed by dipping the capillary end of a capillary pipette into the fluid and drawing the fluid in and out of the pipette several times. The mixture was again centrifuged for ten minutes at the same speed, and the supernatant fluid drawn off to exactly the 0.5 c.c. mark. The sediment was again carefully mixed by using the capillary pipette as described. A drop of this thoroughly mixed suspension was placed in the counting chamber of a standard hemocytometer. The entire ruled area (18 sq. mm.) was inspected with the low power (16 mm.) objective for casts, and the casts were counted and classified. Eight sq. mm. were inspected for cells using the high dry (4 mm.) objective, the cells being differentially counted. The number of elements in twelve hours' urine was calculated as shown by the following example:

*This reagent was $Na_3C_6H_5O_7.2H_2O$ 0.964 gm. plus NaCl 0.589 gm. dissolved in water to a volume of 100 c.c.

Volume of twelve-hour urine	102 c.c.	
Sediment concentration	20:1	
Casts in 1.8 c.mm.	Hyaline	1
	Granular	3
	Total	4
Cells in 0.8 c.mm.	Red blood cells	8
	Epithelial cells and White blood cells	78

From the formula

$$\frac{\text{No. counted} \times \text{Vol. 12 hr. Urine in c.c.} \times 1000}{\text{c.mm. inspected} \times \text{concentration of sediment}} = \text{No. in twelve hours' urine.}$$

The casts were calculated as 11,300; the red blood cells as 51,000; and the epithelial cells and white blood cells as 495,000.

This procedure resulted in the white cells staining varying shades of blue whereas the erythrocytes appeared a yellowish green. Such differential staining resulted in a more accurate differentiation of the white and red cell sediment. The red and white cell casts were similarly differentially stained even though there was complete destruction of the cell morphology within the casts. In addition to the increased facility of counting the cells, the addition of dye and sodium citrate solutions increased the length of time during which the cells and casts remained intact in the specimen.

Table I presents figures showing the agreement between several counts from the same urine and the preservative action of the dye and citrate solutions.

RESULTS

The results obtained in examining the urinary sediments of normal children, children with acute infections, and children with Bright's disease are presented in Tables II, III and IV. Where specimens are obtained without catheterization, as done here and in the usual routine, the epithelial cell count of female children is often increased by the presence of vaginal squamous epithelial cells. We have, therefore, noted the sex of each patient, and whether any vaginal discharge was present.

Table II presents twelve-hour sediment counts of urine obtained from sixteen so-called normal children. For convenience in presenting the data the figures given in the tables are thousands. The group is composed of children admitted to the hospital for pathologic conditions outside the urinary tract, who had had no recent infection or febrile reaction, and who presented no evidence of abnormal renal structure or function. The results are presented so that those of males and females are readily differentiated and the average findings for each sex are separately tabulated. We have also included in the table for purposes of comparison the average normal figures and the maximum limits of

TABLE I

SEDIMENT COUNTS MADE ON SAMPLES FROM THE SAME URINE SPECIMEN USING (1) SODIUM CITRATE CRESYL BLUE, (2) SODIUM CHLORIDE CRESYL BLUE, AND (3) CONCENTRATED URINE UNSTAINED; AND COUNTS MADE AFTER URINE SAMPLES PREPARED BY THE THREE METHODS HAD STOOD DURING SEVERAL DAYS. THE FIGURES REPRESENT THE ACTUAL NUMBER OF ELEMENTS COUNTED IN THE COUNTING CHAMBER.

URINE SPECIMEN	AGE OF SPECIMEN	RED BLOOD CELLS*			EPITHELIAL AND WHITE BLOOD CELLS			CASTS†		
		(1) Na CITRATE DYE	(2) NaCl DYE	(3) CONC. URINE UNSTAINED	(1) Na CITRATE DYE	(2) NaCl DYE	(3) CONC. URINE UNSTAINED	(1) Na CITRATE DYE	(2) NaCl DYE	(3) CONC. URINE UNSTAINED
I	Fresh	1190	1068	1174	64	66	70	26	23	25
I	After 48 hours on ice	1150	1012	1106	58	63	62	24	22	20
I	After 96 hours on ice	1186	977	1008	49	53	60	22	21	25
II	Fresh	393		433	63		66	13		18
II	After 24 hours roo.n temp.	396		291	58		59	16		13
II	After 48 hours room temp.	385		84	62		46	14		7

*8/250 c.mm. counted.

†0.4 c.mm. counted for specimen I and 0.9 c.mm. counted for specimen II.

TABLE II

URINE SEDIMENT COUNTS OF NORMAL CHILDREN

CASE	AGE YEARS	REASON FOR HOSPITAL ADMISSION	12-HOUR URINE VOLUME	SPECIFIC GRAVITY	REACTION LITMUS	ALB.	R.B.C. THOUSANDS	W.B.C. AND EP. CELLS THOUSANDS	CASTS THOUSANDS	PER CENT TYPE OF CASTS
M.P.	7 5/12	Cerebellar tumor	89	1.026	Ac.	0	76.0	1,150.0	34.0	Hyl. 60 Gran. 40
M.G.	10 6/12	Hemi chorea	238	1.020	Ac.	0	208.0	3,659.0	19.8	Hyl. 33 Gran. 67
G.O'H.	5 8/12	Chronic poliomyelitis	102	1.032	Ac.	0	51.0	495.0	11.3	Hyl. 25 Gran. 75
A.M.	7	Cerebral palsy	60	1.025	Ac.	0	21.0	635.0	14.6	Hyl. 100
H.M.	8 2/12	Cerebral palsy	140	1.032	Ac.	0	284.0	2,750.0	19.4	Hyl. 75 Gran. 25
A.B.	5 9/12	Anal fissure; eczema	87	1.037	Ac.	0	374.0	2,800.0	0.0	
G.S.	7 6/12	Thrombocytosis; ? Mesenteric thrombosis	194	1.025	Ac.	0	123.0	1,484.0	3.2	Gran. 100
M.H.	8 6/12	? Petit mal	206	1.027	Ac.	0	306.0	4,106.0	17.0	Hyl. 50 Gran. 50
Ave. Girls	7 7/12		142	1.028	Ac.	0	180.4	2,160.0	14.8	Hyal. 49 Gran. 51
T.C.	8 11/12	Epilepsy	178	1.027	Ac.	0	157.0	556.0	32.6	Gran. 100
B.M.	8 10/12	Night terrors; ? Brain tumor	162	1.032	Ac.	0	75.0	637.0	0.0	
V.S.	3 11/12	Cerebellar tumor	154	1.024	Ac.	0	84.2	716.0	10.7	Hyl. 100
S.D.	4 1/12	Cerebral palsy	99	1.028	Ac.	0	70.0	366.0	10.4	Hyl. 33 Gran. 67
E.S.	4	Eczema	77	1.030	Ac.	0	72.0	498.0	2.7	Hyl. 100
W.R.	6 1/12	Mild encephalitis	149	1.030	Ac.	0	70.0	605.0	15.5	Hyl. 100
P.	9 6/12	? Oxycephalus	170	1.027	Ac.	0	106.0	833.0	23.5	Hyl. 25 Gran. 75
C.S.	3 6/12	Mental Retardation; ? Amyotonia	64	1.032	Ac.	0	60.0	840.0	37.8	Hyl. 74 Gran. 26
Ave. Boys	6 2/12		157	1.029	Ac.	0	86.8	631.0	16.6	Hyl. 62 Gran. 38
Ave. 16 Cases	6 10/12		149.5	1.028	Ac.	0	133.6	1,395.0	15.7	Hyl. 55 Gran. 45
Ave. 74 Med. Stu. Under 30 (Addis)			381	1.032	Ac.		65.7	322.5	1.04	Hyl.
Upper limits of normal (Addis)							425.0	1,835.0	4.27	
Ave. 44 Med. Stu. Under 30 (Goldring)			289	1.030			146.0	540.0	1.3	Hyl.
Upper limits of normal (Goldring)							1,530.0	3,400.0	9.2	

TABLE III

URINE SEDIMENT COUNTS FROM PATIENTS WITH ACUTE INFECTIONS

CASE	AGE IN YEARS	DIAGNOSIS	HIGH TEMP.	TEMP. AT TIME OF COUNT	12-HOUR URINE VOLUME	SPECIFIC GRAVITY	REACTION	ALB. DURING FEVER	ALB. IN SPEC.	RED BLOOD CELLS THOUSANDS	WHITE BLOOD AND EPITHELIAL CELLS THOUSANDS	CASTS THOUSANDS	PER CENT TYPE OF CASTS
R.R.	5 5/12	Primary pneumonia	105.0	99.0	94	1.031	Ac	VST	0	590.0	1,056.0	8.3	Hyl. 20 Gran. 80
G.W.	7 4/12	Acute pansinusitis	103.2	100.2	118	1.030	Ac	0	0	263.0	1,840.0	4.1	Hyl. 50 Gran. 50
M.P.	8 6/12	Facial erysipelas	105.0	100.0	175	1.027	Ac	SPT	0	1,040.0	4,620.0	286.0	Hyl. 28 Gran. 72
R.Q.	3 6/12	Otitis media bilat.; primary pneumonia	105.0	99.8	132	1.031	Ac	0	0	268.0	1,580.0	22.9	Hyl. 42 Gran. 58
M.W.	6 2/12	Primary pneumonia	*105.0	100.0	146	1.035	Ac	SPT	0	416.0	5,760.0	10.1	Hyl. 75 Gran. 25
C.C.	4 5/12	Nasopharyngitis	102.6	99.2	38	1.028	Ac	ST	SPT	90.0	470.0	2.6	Hyl. 50 Gran. 50
M.E.	3 1/12	Otitis media bilat.; primary pneumonia	104.2	99.0	118	1.021	Ac	0	0	626.0	4,100.0	8.2	Hyl. 50 Gran. 50
J.F.	2 8/12	Sinusitis; nasopharyngitis	104.0	104.0	115	1.022	Ac	SPT	SPT	323.0	2,290.0	12.0	Hyl. 50 Gran. 50
Ave. Girls	5 3/12				117	1.028				452.0	2,714.0	31.8	Hyl. 47 Gran. 53

*Count made nine days from peak of temperature and seven days from temperature at 104° F.

TABLE III—CONT'D

CASE	AGE IN YEARS	DIAGNOSIS	HIGH TEMP.	TEMP. AT TIME OF COUNT	12-HOUR URINE VOLUME	SPECIFIC GRAVITY	REACTION	ALB. DURING FEVER	ALB. IN SPEC.	RED BLOOD CELLS THOUSANDS	WHITE BLOOD AND EPITHELIAL CELLS THOUSANDS	CASTS THOUSANDS	PER CENT TYPE OF CASTS
C.T.	6 1/12	Primary pneumonia	105.0	99.6	312	1.027	Ac	0	0	202.0	460.0	9.2	Hyl. 50 Gran. 50
G.F.	2 11/12	Primary pneumonia	103.2	100.6	96	1.028	Ac		0	206.0	570.0	78.0	Hyl. 58 Gran. 28 Fatty 14
C.H.	3 5/12	Rheumatic fever; pneumonia	104.2	99.6	206	1.024	Ac	ST	0	181.0	1,350.0	5.4	Gran. 100
J.C.	5 6/12	Primary pneumonia; empyema	104.8	104.8	308	1.015	Ac	0	0	650.0	4,740.0	7.4	Gran. 100
C.C.	6 7/12	Otitis media rt.; primary pneumonia	105.0	100.2	91	1.028	Ac	SPT	0	77.0	780.0	6.2	Hyl. 50 Gran. 50
F.H.	3 10/12	Otitis media bilat.; primary pneumonia	105.0	99.0	103	1.025	Ac	0	0	162.0	2,060.0	62.6	Hyl. 95 Gran. 5
C.T.	2 1/12	Otitis media lt.; acute upper resp. inf.	103.0	99.0	45	1.028	Ac	T	0	200.0	480.0	5.6	Hyl. 75 Gran. 25
G.M.	4 4/12	Bronchopneumonia	103.0	99.8	114	1.027	Ac		0	668.0	855.0	11.9	Gran. 100
Ave. Boys	4 4/12				159	1.025				293.0	1,412.0	23.2	Hyl. 41 Gran. 59
Ave. 16 cases	4 10/12				139	1.026				372.0	2,063.0	27.5	Hyl. 44 Gran. 56
Ave. 70 counts on 16 rheumatic fever cases (Goldring)				99-101	437	1.028				857.0	3,360.0	5.32	Hyl. 80 Gran. 19
Ave. 16 counts on rheumatic fever cases (Goldring)				101-105						3,810.0	56,340.0	495.0	Cell. 1
Ave. 48 counts on 37 pneumonia cases (Goldring)				102.4						520.0	3,036.0	550.0	

normal observed in adults by Addis[11] and Goldring.[13] The presence or absence of albumin as determined by the nitric acid contact ring test is also recorded.

Table III presents twelve-hour sediment counts on urines obtained from patients with or just following acute infections. In this table we have included columns showing the maximum temperature preceding the count and the temperature at the time the count was made. With the exceptions noted in the table the maximum temperature occurred five days or less before the count. We have also divided the albumin column into two parts. In the first column under albumin is recorded the absence or presence of albumin by the heat and acetic acid test during the febrile period, and in the second column under albumin is recorded the absence or presence of albumin at the time the urine specimen was collected. Here again the two sexes have been grouped separately and averages for each sex tabulated. We have also tabulated the average figures found by Goldring for adult patients with rheumatic fever[14] and pneumonia.*[13]

Table IV presents the findings in six cases of Bright's disease. They are presented here in order to give a comparison of the figures in patients with Bright's disease with the figures of groups one and two.

DISCUSSION OF RESULTS

Examination of Table II shows that the normal upper limit for red blood cell counts in twelve-hour urine established by Addis for adults would seem to apply to both male and female children for specimens collected without catheterization. The average girls' red blood cell count of 180,400 cells does not differ significantly, considering the range of normal variability, from the average boys' red blood cell count of 86,800 cells. No red blood cell count among the girls exceeded Addis' upper limit of normal of 425,000 cells. None of our figures from the normal group approached Goldring's upper normal limit of 1,500,000.

The counts of epithelial and white blood cells would, however, seem to indicate a higher normal figure for girls than for boys. This is perhaps as might be expected where the urine specimens are collected without catheterization. Four of the eight epithelial and white cell counts from girls fall above Addis' upper limit of normal male adults, whereas all the counts from boys fall within his normal range. Considering our limited number of boy cases our average male epithelial and white cell count of 631,000 agrees very well with Addis' average of 322,500 and Goldring's average of 540,000. Our data would indicate an epithelial and white blood cell count of 4,500,000 as an approximate

*In calculating the average figures for Goldring's pneumonia cases, we have omitted seven of the forty-four cases; namely Cases 1, 2, 12, 17, 20, 31, and 44. These cases had evidence of renal or urinary tract disease.

TABLE IV

TABLE IV

URINE SEDIMENT COUNTS FROM PATIENTS WITH BRIGHT'S DISEASE

CASE	TYPE OF BRIGHT'S DISEASE	12-HOUR URINE VOLUME	SPECIFIC GRAVITY	REACTION	ALBUMIN	RED BLOOD CELLS THOUSANDS	WHITE BLOOD AND EPITHELIAL CELLS THOUSANDS	CASTS THOUSANDS	PER CENT TYPE OF CASTS	
M.C.	Acute hem.	185	1.022	Ac.	T	451,000.0	25,900.0	787.0	R.B.C.	40
									Hyl.	32
									Gran.	16
									Fatty	10
									R.F.	2
J.G.	Chronic active hem.	470	1.021	Ac.	LT	40,000.0	128,000.0	6,700.0	Hyl.	81
									Waxy	19
									R.B.C.	> 1
R.P.	Initial hem.	225	1.017	Ac.	ST	166,000.0	5,910.0	156.0	R.B.C.	40
									Hyl.	20
									Gran.	20
									Fatty	15
									R.F.	5
J.T.	Initial hem.	220	1.020	Ac.	T	398,000.0	30,000.0	200.0	R.B.C.	40
									Fatty	40
									Gran.	20
M.S.	Initial hem.	45	1.012	Ac.	LT	405,000.0	52,000.0	128.0	R.B.C.	29
									Hyl.	43
									W.B.C.	14
									Gran.	14
R.J.	Degenerative (lipoid nephrosis)	112	1.026	Ac.	LT	232.0	4,080.0	318.0	Hyl. with fat drops	95
									Waxy	5
(Addis) Ave. Typical	Initial hem.	238	1.020	Ac.	T	1,500,000.0*	35,000.0	200.0	R.B.C.	25
									Epith.	11
									Fatty and waxy	10
									Gran.	17
									Hyl.	37
(Addis) Ave. Typical	Chronic active hem.	425	1.016	Ac.	T	110,000.0†	30,000.0	200.0	Hyl.	55
									R.B.C.	5
									Gran.	10
									Waxy and fatty	5
									R.F.	25
(Addis) Ave. Typical	Degenerative	350	1.023	Ac.	LT	0 to few	16,000.0	630.0	Hyl.	90
									Fatty and waxy	8
									Gran.	2

* ± 1,000,000,000.0 cells. † ± 100,000,000.0 cells.

upper limit of normal for twelve-hour specimens collected without catheterization from girls. This figure is not very much greater than Goldring's normal upper limit for males of 3,400,000. In the absence of better evidence Addis' upper limit of 1,800,000 may be taken as the upper limit of normal epithelial and white blood cell counts for twelve-hour urine from boys, for we have no figure approaching Goldring's high normal limit.

In examining the twelve-hour urine cast counts one finds no significant difference in those for boys and for girls. The average, however, of the two groups, namely 15,700 casts, is markedly higher than Addis' and Goldring's average of 1,170, and the two highest counts of 34,000 and 37,800 are appreciably greater than the higher of their upper limits, 9,200 casts. This difference may be due to the more rapid desquamation of renal cells in the child as compared with the adult. There is the added difference of differential cast count. In the normal adult the casts are approximately 100 per cent of the hyaline type. The cases, both boys and girls, of Table II, show the fairly consistent occurrence of granular casts, the average differential being 55 per cent hyaline and 45 per cent granular. The majority of the casts of both types were of a very small size. Our data would indicate an average normal cast count in a twelve-hour specimen of approximately 16,000,

TABLE V

GROUP	SEX	NO. OF CASES	12-HOUR URINE VOLUME	SP. G. OF URINE	RED BLOOD CELLS THOUSANDS	WHITE BLOOD AND EPITHELIAL CELLS THOUSANDS	CASTS THOUSANDS
Upper limits normal girls	F	8	238	1.037	274.0	4,106.0	34.0
Upper limits normal boys	M	8	178	1.032	157.0	840.0	37.8
Upper limits normal (Addis)	M	74			425.0	1,835.0	4.27
Upper limits normal (Goldring)	M	45			1,530.0	3,400.0	9.2

applicable to both girls and boys, and an upper limit of normal of 40,000 casts. Thus the cast count of normal children and normal adults may be of a very different order of magnitude. And in determining the presence of Bright's disease according to Addis' criteria, the normal range of casts must be changed from 5,000 to 40,000 per twelve hours.

In using sediment counts for diagnostic purposes, comparison of a given count with the upper limit of normal is perhaps of more significance than comparison with the average normal figure. For convenience we have summarized in Table V the upper limits of the counts from our normal children and those given by Addis and Goldring in their series of normal men.

In examining the sediment counts from children with or immediately following acute infections one finds five of the sixteen red blood cell counts above 425,000 but no figure higher than Goldring's upper limit of normal of 1,500,000, our highest count in this group being 1,040,000.

Three of the sixteen cases, two girls and one boy, have epithelial and white cell counts above 4,500,000; that is, above the upper limit of normal for girls. The counts in the two girl patients are only slightly above this upper limit, and many squamous epithelial cells were present in both specimens of urine. These then cannot be considered as significant increases. The count of 4,740,000 in the boy patient, Julius C., is significantly higher than Addis' upper limit of normal, but only slightly higher than the figure of 3,400,000 reported by Goldring.

Three of the sixteen cases had cast counts above 40,000. The only case where the cast count was markedly increased was in a girl, Marie P., suffering from erysipelas. It will also be noted that this was the only case of the three in which an increase in the cellular elements accompanied the increase in casts.

The increase in the averages of the counts of Table III as compared to the averages of Table II would seem to indicate an increase in the urinary sediment in the acute infection group above the normal averages. The considerations of the previous paragraphs, however, show that no great significance can be attached to increases of this order of magnitude. The examination of the counts presented in Table IV gives further evidence for such a conclusion.

As already mentioned, the counts of Table IV on cases of Bright's disease are reported here in order to give an idea of the order of magnitude of the increase in cellular elements accompanying real kidney pathology. The counts were obtained from five patients with hemorrhagic Bright's disease and one patient with degenerative Bright's disease or lipoid nephrosis. Three of the hemorrhagic cases were of the initial type and stage of the disease; one was a patient presenting an exacerbation following an initial stage, and we have followed the precedent of Peters and Van Slyke[15] in designating this as "acute" rather than "initial"; and one of the hemorrhagic cases was in the chronic, active stage of the disease. As the counts on these patients agree with Addis' counts from patients suffering with the same type and stage of the disease, they have a significance greater than could otherwise be attached to so limited a number of cases. If we include a rough quantitative estimation of urinary albumin with the consideration of the urinary sediment, there is a marked difference between the urinary findings in the acute infection group and the Bright's disease group. We wish to call attention to the appearance of casts in this group not found in the normal or acute infection groups. In all the cases of Table IV we find blood cell, waxy, or renal failure casts.[16]

In the cases of initial or acute hemorrhagic Bright's disease the lowest red blood cell count is 166,000,000 compared to the upper normal limit of 425,000 in Addis' cases and 1,500,000 in Goldring's cases. In the one case of chronic active hemorrhagic nephritis the red blood cell count is 40,000,000. In terms of such figures the maximum red blood cell count in the acute infection group (Table III) of 1,040,000 cells might be considered within the normal range. The one possibly significant epithelial and white blood cell count on Julius C. (Table III), should probably be considered insignificant in view of the normal red blood cell and cast counts. In comparing the cast counts of Tables III and IV it would seem that of the former the count of Marie P. could alone be considered as indicative of renal damage.

Table III also shows that of the sixteen cases with acute infection eight showed albumin in the urine during the infection and within four days of the time of making the sediment counts. Of these eight, only one (Marie P.) had a sediment count suggestive of structural renal damage. In this one case the evidence was not convincing.

None of the patients with acute infection had evidence of dehydration or retention at the time the counts were made. With proper therapy both dehydration and retention can usually in such conditions as included in our cases be kept within what might be termed physiologic limits. Our data, therefore, are to be considered as representing the findings in urine sediments during or immediately following acute infections without the presence of dehydration. Had dehydration been present the results might have been quite different. The work of Schiff and Bayer,[17] as well as that of Schloss,[1] indicates that dehydration may have a greater effect than infection on altering the urinary sediment.

The data reported in this paper would seem to indicate that during the course of many acute infections there is no significant increase in the urinary sediment that can be correlated with the infection per se. We know that dehydration accompanying an acute infection may result in the retention of nitrogenous substances due to the functional impairment of kidney function. Possibly dehydration produces changes in the function or structure of the kidney that alter the urinary sediment. The use of quantitative sediment counts in examining the urine of dehydrated subjects will aid in eliminating the uncertainty of the impression recorded by previous workers. The data so obtained will contribute to the soundness of Addis' opinion concerning the relation between urine sediment and the presence of a renal lesion. The pathologist[1] has found little evidence of structural change.

The data given here have not answered the question presented in the introduction, but they do indicate that acute infection as such can be eliminated from the problem. In doing this, no matter what the find-

ings in dehydration may be, the data have a direct bearing on the indicated treatment during the course of most acute infections. For with nonnephritic patients we can assume, until proved otherwise, that any sign of kidney insufficiency as interpreted by retention of nitrogenous substances in the blood or of kidney irritation as interpreted by urinary sediment is not the result of renal damage from the existant infection, but is an indication of the existence of conditions within the body unfavorable to adequate kidney function. With the attention directed toward these abnormal conditions (i.e., increased catabolism and dehydration) their detection and correction are usually readily accomplished.

SUMMARY

A method of differentially staining white blood and epithelial cells from red blood cells in urinary sediment in a counting chamber is described. This method has increased the accuracy of our qualitative urine sediment counts.

The urine sediment counts on sixteen normal children, sixteen children with acute infections, and six children with Bright's disease have been presented.

The occurrence of a higher count of casts in the twelve-hour urine of normal children than that in normal adult males is mentioned.

No significant increase in the urinary sediment was observed in the children with acute infections.

In the discussion we have called attention to the necessity of distinguishing between the effects of infection per se and dehydration. The bearing of the reported data on the treatment of acute infections is discussed.

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EPIDURAL ABSCESS COMPLICATED BY STAPHYLOCOCCIC MENINGITIS

Report of a Case With Complete Recovery Following Operation*

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SCHMALZ[1] in 1925 collected 43 cases of acute epidural infection from the literature and cited 3 operations with one recovery. Pineoffs[2] in 1926, cited one case with operation and recovery and reported 3 additional cases with one recovery. Dandy[3] in 1926 collected 25 cases from the literature and summarized them in the following words:

"Although all the acute infections have run a fulminating and fatal course, except two that were drained, it is hoped that with an early and accurate diagnosis, operative treatment (drainage) may be effectual in saving both life and function."

A number of cases have since been reported, and at the present time, while the disease is relatively infrequent, epidural abscess is considered a distinct disease entity, with a definite symptomatology that sometimes permits of accurate diagnosis and successful treatment. However, only a few recoveries have been reported, and in nearly all of these there have remained paralyses and serious dysfunction. A case of primary epidural abscess, complicated by a septic meningitis, with complete recovery, seems therefore worth reporting.

On July 22 a white girl aged six and one-half years was brought for an x-ray examination because her mother thought she had swallowed a pin. She complained of a pain in the left loin, but there was no tenderness. The x-ray revealed no foreign body in the intestine or any spinal pathology, and the physical examination and routine laboratory tests at this time were negative. The following day she had a slight fever and the pain in the back was constant, but not severe enough to stop the child from play.

On July 24 there was little change in her condition, the pain was constant and sometimes the child cried with it and said it radiated down the left hip and leg. She walked on her toe because it hurt her back less to walk this way. July 25 I was called to the home because the pain had increased and she had developed fever. The child did not appear very ill, but the temperature was 103° F. The back pain was constant, and the left leg was drawn up and it hurt to straighten it. The neck was a little spastic, and Brudzinski's sign was very slightly positive. There was marked pain on bending the spine forward.

Chloroform anesthesia was given and 15 c.c. of clear spinal fluid under very slightly increased pressure was withdrawn. The fluid contained 17 cells, all lymphocytes, no globulin and no organisms. A tentative diagnosis of poliomyelitis

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was made and the child was removed to the isolation hospital where she was given 125 c.c. of whole blood from the father, no immune serum being immediately available.

The family history was unimportant and the child had had no illness except chicken pox, a mild attack of tonsillitis during the winter and furunculosis two years before. The physical examination was essentially negative. The neurologic examination showed a diminished patella reflex on the left side, somewhat increased abdominal reflexes below the umbilicus, a slightly positive bilateral Kernig, positive Brudzinski, and moderate hyperesthesia over the left loin region. There was no tenderness over the vertebrae, except between the fourth and fifth lumbar where the puncture was done. Her blood showed 5,480,000 R.B.C.; 16.1 gm. Hg; 7,280 W.B.C.; 70 per cent polys; 25 per cent lymphocytes; 5 per cent unclassified. The blood Wassermann and Kahn tests were negative as was the spinal fluid Wassermann, Kahn, and Kline tests. The urine was negative.

On July 26 the patient awakened with a temperature of 99° F. and said she felt fine. A marked bilateral Kernig was the only change in her physical findings. She had a good day but the temperature rose to 100° F. in the afternoon and at 6:30 P.M. she went to sleep. At 7:25 P.M. she was found in coma, and could not be roused. Her pulse was 160 and respiration 22. She lay on her left side with knees drawn up, neck retracted, and back arched. The eyes were rolled back and pupils widely dilated. Lumbar puncture was done, under ethylene anesthesia, and a plug of pus was followed by slightly cloudy fluid under terrific pressure. Forty-five c.c. of fluid were withdrawn and 10 c.c. concentrated antimeningococcic serum were given through the same needle. The spinal fluid contained 60 cells, all lymphocytes, the globulin was negative and no organisms were found on smear. Cultures were made and a blood culture taken. At 8:30 she roused and the pulse dropped to 80.

The following day the spinal fluid culture was reported positive for Staphylococcus aureus. Lumbar puncture was done at 8- to 12-hour intervals, about 50 c.c. of fluid being recovered each time. The spinal fluid at this time contained 4,220 cells, 75 per cent being polynuclears. The globulin was 4-plus positive and smears showed large numbers of gram-positive organisms. The spinal fluid sugar was 90 mg. per 100 c.c. and the chlorides 720 mg. per 100 c.c. The white blood count was 11,000 with 81 per cent polys. The temperature remained around 103° F. all day and the sensorium was clear.

On July 28 she had an uneventful day, 60 c.c. of fluid being removed by puncture at noon. At 9 P.M. she had a pulse of over 200 per minute and signs of increased intracranial pressure. Sixty-five c.c. of fluid were removed and the pulse rate slowly fell.

On the morning of July 29 the spinal puncture needle was introduced between the third and fourth lumbar vertebrae. Before the needle punctured the dura a stream of thick yellow pus escaped, followed by cloudy spinal fluid as the needle was shifted. This procedure was repeated, introducing the needle between the second and third lumbar vertebrae. Again pus was encountered, though in smaller quantity, followed by spinal fluid when the needle snapped through the dura. The pus contained 7 to 10 clumps of staphylococci to the oil immersion field.

A diagnosis of epidural abscess with a secondary meningitis seemed now conclusive, and immediate operation was agreed on.

Dr. Eustace Semmes concurred in the diagnosis and with ethylene anesthesia did a laminectomy. Yellow pus welled into the wound from beneath the third lumbar vertebra, about one ounce escaping. Because of the complicating meningitis the dura was opened and a flare end 16 French catheter sutured inside the dural flaps. A second catheter was left extradurally to drain the abscess cavity, which was irrigated with Dakin's solution and saline. Enormous quantities of fluids were

given to increase drainage. A spectacular drop in temperature and pulse followed the operation. The wound drained freely and on August 3 the drain was removed from the abscess cavity. Cramping pains in the leg were complained of. The blood at this time showed 28,280 W.B.C. with 75 per cent polys and the blood culture was negative. Serum sickness developed on August 4 and on August 9 the wound was reopened to secure better drainage.

Recovery was uneventful and the patient was allowed to go home on August 16. A spastic deformity which lasted several months gradually cleared up following the final healing of the wound.

This case is interesting in that it was a hematogenous infection; it was primary; it was in the lumbar region; and there was only one abscess. The meningitis was undoubtedly caused by carrying the infection into the dural envelope with the puncture needle. An unusual feature was the unilateral character of the pain and the absence of a cord level in the sensory symptoms. In nearly all reported cases this has been sharply defined, though the rarity of the condition gives us little familiarity with its clinical characteristics.

The mortality from septic meningitis has been reduced about 50 per cent by surgical drainage and forced fluids, but recovery from both epidural abscess and septic meningitis is both interesting and unusual. Dr. Semmes has operated on one other case of epidural abscess with complete recovery which I here wish to note.

CONCLUSIONS

A case is reported of primary, hematogenous epidural abscess localized in the lumbar region. Cerebrospinal staphylococcic meningitis developed after the introduction of the spinal puncture needle through the infected area. Complete recovery followed laminectomy and drainage of the abscess cavity, plus an intradural drain and the administration of large quantities of fluid.

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MADISON AVENUE AT SOMERVILLE.

CONGENITAL CYSTIC DISEASE OF THE LUNG*†

Report of a Case

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CONGENITAL cystic lung disease was first described by Meyer[1] in 1859. Koontz[2] in 1925 collected 108 cases and reported an additional case. Since that time there have been a number of cases reported, particularly in the German and French literatures, but the rarity of the condition makes it seem advisable to report the occurrence of each additional case.

The following is the first case of cystic lung disease to be seen in this clinic, although we have on two occasions seen cases of solitary mediastinal cysts with clinical pictures very similar to that seen in the former condition:

P. G., a male infant of Armenian parentage, aged five and one-half months, was admitted to the hospital February 13, 1931, with the complaints of cough, difficult breathing and swelling of the abdomen.

Six weeks before entry the child developed a cough and slight fever and was thought to have a "cold." Shortly thereafter he was seen in the Out-Patient Department because of difficulty in breathing and the persistence of cough. Examination revealed that respiration was accompanied by retraction of the diaphragmatic attachment, that the left chest was tympanitic with distant breath sounds, that the heart was displaced to the right, and that the spleen was enlarged. Admission to the hospital was refused by the parents, and during the following five weeks the infant had intermittent periods of fever and labored breathing and persistent cough. On February 11, 1931, he vomited several times, developed swelling of the abdomen and a high fever, and was admitted to the hospital two days later.

When admitted he was critically ill, with a temperature of 103° F. The respirations were rapid and shallow. The thorax moved but little with respiration, and the two sides seemed to expand equally. The left chest was tympanitic except at the base posteriorly where resonance was impaired. The breath sounds were distant throughout the left lung. There were exaggerated compensatory breath sounds throughout the right lung. The abdomen was distended with shifting dullness present, but was nontender. The spleen was enlarged, firm, and extended two finger-breadths below the costal margin. The scrotum was reddened and there was a left hydrocele.

Laboratory Data.—The urine was normal. The blood hemoglobin was 70 per cent. There were 6,000,000 erythrocytes per c.mm. The leucocytes numbered 39,000 per c.mm. The differential white cell count showed 76 per cent neutrophils, 15 per cent myelocytes, 8 per cent lymphocytes and 1 per cent monocytes. The

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†Submitted for publication, May 28, 1932.

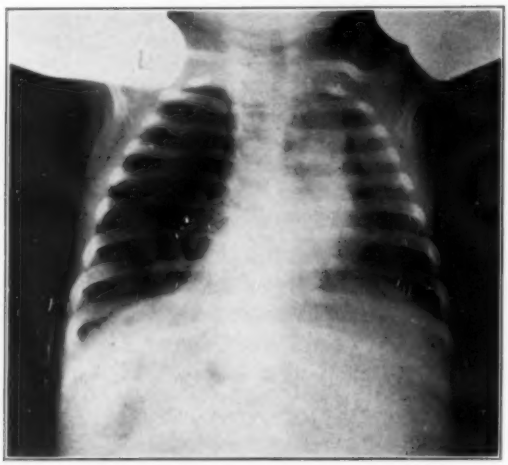

Fig. 1.—Roentgenogram showing the emphysematous left lung with the heart and mediastinum displaced to the right.

Fig. 2.—Showing the extreme emphysema in the cystic upper lobe of the left lung.

Wassermann reaction was negative. The tuberculin test was negative. Hemolytic streptococci were cultivated from the blood.

Roentgenograms (Fig. 1).—The left lung was emphysematous. The heart and mediastinal contents were displaced to the right. There was a considerable amount

Fig. 3.—Low power photomicrograph showing parts of several large cystic cavities and the emphysematous alveoli of the left upper lobe.

Fig. 4.—High power photomicrograph showing the nature of the walls of the cystic cavities and the lining columnar epithelium.

of rather discrete peribronchial thickening on both sides. Fluoroscopy showed the diaphragmatic movements to be limited on both sides, but the left diaphragm moved less than the right and was low and flat. These findings were interpreted by

Dr. E. C. Vogt as suggesting incomplete obstruction of the left main bronchus with generalized bronchitis. The presence of a foreign body had to be considered.

Course.—Following admission the breathing became more labored, cyanosis developed and physical signs suggesting infiltration of the right lung appeared. Therapy directed toward relieving the abdominal distention failed. There was a progressive increase in the scrotal swelling. Bronchoscopy was not done because of the patient's critical condition. The temperature remained high. The hippocratic facies became evident on the third day, at which time the peritoneal cavity was drained by incision under local anesthesia. A moderate amount of thin, purulent exudate was obtained from which hemolytic streptococci were cultured. The infant's condition became progressively worse and death occurred three days after admission to the hospital.

Necropsy.—On removing the chest plate, the heart and pericardial sac were completely hidden from view by the markedly emphysematous upper lobe of the left lung (Fig. 2). The heart and mediastinum were displaced to the right. The left lung had two well-defined lobes, and the upper lobe presented on its inferior surface a small accessory tab of lung tissue. The surface of the left upper lobe showed numerous air-containing blebs. Just below the bifurcation of the trachea an underdeveloped bronchus with soft, collapsible walls, measuring 3 mm. in diameter, was given off. This seemed to contain fewer cartilaginous rings than normal, and when followed into the left upper lobe, appeared to end abruptly in the emphysematous lung tissue. The left main bronchus, just before passing into the lower lobe, gave off a small branch which curved abruptly and passed into the base of the upper lobe. The left lower lobe was firm and pneumonic, and the right lung showed a fibrinopurulent exudate on the surface and partial consolidation of all lobes. In addition there was an acute peritonitis, acute mesenteric adenitis, acute splenitis, and acute hepatitis.

Microscopic Examination.—Sections of the left upper lobe (Figs. 3 and 4) showed all the bronchi and alveolar spaces to be markedly dilated, giving the impression under low power of numerous cysts, some lined by flattened alveolar, others by cuboidal and columnar epithelium, the latter being the bronchi. Cilia were noted in the columnar cells lining some of the spaces. The bronchial structures were striking because of their large number, the degree of dilatation, and papillary infoldings. In some areas the dilated bronchioles were about as numerous as were the alveoli. In the sections no actual obstruction to the bronchi could be demonstrated. In appropriate places the dilated bronchi could be distinctly traced into distended ampullae and terminal air sacs.

DISCUSSION

Congenital cystic disease of the lung occurs in two forms, one the solitary cyst, and the other a diffuse, cystic honeycombing of part or all of a lung. In the former type symptoms may not occur until late in life and then are usually dependent upon the development of a pulmonary infection which leads to an examination which discloses the presence of one or several cysts. In the latter type, of which this case is an example, the symptoms usually develop early in infancy and should cause one to consider the possibility of congenital cystic lung disease.

In the infantile form, unexplained attacks of dyspnea and cyanosis with the signs of pneumothorax, i.e., hyperresonance and greatly diminished breath sounds on the affected side, and exaggerated breath sounds and displacement of the heart and mediastinum to the opposite side, should cause one to think of cystic lung disease. Especially significant are these signs when the past history discloses recurrent periods of labored breathing and when no evidence of pulmonary infection or trauma is present to explain the development of a pneumothorax. The roentgenograms will in some cases show a honeycombed appearance of the affected lobe, and in nearly all cases enough lung markings present to exclude pneumothorax. In those cases, of which the one above described is an example, where the cystic cavities are so small as not to throw a shadow in the roentgenogram, partial bronchial obstruction must be considered to be present and the presence of a foreign body excluded by the history and by bronchoscopy in order to arrive at the correct diagnosis. Having so far as possible excluded other possibilities, the diagnosis may definitely be established, as suggested by Eloesser,[4] by the injection of lipiodol into the lung tissue through the chest wall, after which a roentgenogram will show the cystic dilatations. Such a procedure is not particularly dangerous, as the production of a pneumothorax may be of decided benefit to the patient.

That cystic lung disease is of congenital origin seems well established. It is more difficult to explain the sudden appearance of symptoms and in some cases their intermittent nature with periods of well-being in the intervals between attacks. Miller[5] believes the attacks of cyanosis and dyspnea dependent upon the rupture of a cortical cyst which is in communication with the bronchial tree, causing a pneumothorax to develop with acute respiratory symptoms and later the spontaneous recovery. This seems unlikely in view of the postmortem and clinical finding of an emphysematous lung at the time of the acute respiratory symptoms in the case described above and in other reported cases. It would seem more likely that malformed bronchi with readily collapsible walls, as seen here, might become partially obstructed by increasing pressure within the thorax, allowing a valvelike action with the ingress but not the egress of air up to a point where rupture of a cyst occurs with temporary pneumothorax and improvement in the clinical condition.

SUMMARY

A case of congenital cystic lung disease with the clinical, roentgenologic and pathologic findings of partial bronchial obstruction is reported. A brief description of the findings in the disease is given, and a possible explanation for the symptoms offered.

I am indebted to Dr. George M. Haas for the photomicrographs and to Dr. E. C. Vogt for the roentgenologic interpretations in this case report.

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300 LONGWOOD AVENUE.

EXOPHTHALMIC GOITER IN IDENTICAL TWIN GIRLS*†

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THE occurrence of embryonic duplication in homologous twins has always been a matter of interest.‡ Reports are beginning to be more common in the medical literature showing the tendency for both individuals to undergo the same pathologic changes. It has been known for some time that in multiple fetation there may be insufficient supply of minerals so that congenital anemia, hypocalcemia, and other abnormal states may exist. A few twins have been reported having nervous or mental diseases of similar type, which in some instances were acquired almost simultaneously. Contagious, transmissible diseases and dietary deficiencies would be expected to occur in both individuals if in the same environment; under such a circumstance the failure of the similar twin to get a like disease would be more remarkable. The similarity of development and hereditary characteristics in a set of identical twins, therefore, is no more striking than is their "morbid sympathy in disease" as Shirley Smith[1] has expressed it, both facts showing the congenital influence upon physical appearance as well as susceptibility to the same disease processes.

REPORT OF THE CASES

Following is an abstract of the findings:

Twin sisters, R. and F., were born in 1920, residing until the age of nine years in one of the lake states where goiter is endemic. Birth was premature, the weights 4½ and 4¼ pounds respectively, delivery normal, breast fed for three months, growing rapidly, but not walking until eighteen months, nor talking until two and one-half years. At four years they had chickenpox, at five years measles, several attacks of tonsillitis coincidently, and about August 1, 1931, tonsillectomy was performed. At eight years of age, it was noticed that R. had prominence of the eyes and an enlargement of the neck. Two years later F. developed a noticeable bulging of the eyes and during the same year prominence of the neck. They were active, bright, well-nourished girls. In August, 1931, at the age of ten

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†Read by title at the American Pediatric Society, May 26, 1932.

‡The opportunity was taken with the consent of the mother, while the twins I am reporting were at the hospital, to interchange transplants of a small patch of skin. Permanent tissue transplants are difficult if not impossible to attain in individuals other than identical twins. In this instance, several months later, the grafts were still living. This work was performed by Dr. Earl C. Padgett, Department of Surgery. He states that in single birth individuals he has been unable to make large skin transplants grow, but that in four sets of homologous twins the interchange has been viable four or five months later. It is theoretically possible for transplants from such individuals to remain viable permanently. Dr. Padgett expects to publish his experiments and his hypothesis later, but he feels that for all practical purposes homeotransplantation may be relegated to the realms of medical mythology.

years and eight months, the girls showed a tendency to undue fatigue and a loss of weight, and they were brought into our hospital for observation and treatment.

The father died at fifty years of age of apoplexy; the mother's health is good and without thyroid disturbance; a sister of the twins is well at thirteen years without thyroid enlargement. An aunt has goiter. No tuberculosis or syphilis in the family. That the twins were similar from the beginning is indicated by the fact that the father was never able to tell them apart. The mother admits that she cannot always identify them. There have been only slight differences during their life, at the time of admission the weight varied only three-fourths of a pound, and the height one-half inch.

The similarity of the girls puzzled the hospital attendants. The hospital notes stated that there is similar mouth-breathing, prominence of upper jaw, malocclusion,

Fig. 1.—F and R. Photograph taken after two months' rest in bed.

high arched palate; widened palpebral fissures, tendency to exophthalmos, inability to wrinkle the forehead. R. shows a rather diffuse soft right lobe enlargement of the thyroid, the same lobe in F. was somewhat hard and nodular.

The similarity of their history is shown by the following:

Both children had acute purulent sinusitis on admission which cleared up in three weeks. While this infection was present, each had tenderness and stiffness in the right knee. These girls did not show fine tremors, during the months of our observation. In hypothyroidism of childhood, fine tremors are regarded as unusual.

	R.	*F.*
Height	58¼ in. (149.95 cm.)	58¾ in. (149.22 cm.)
Pulse on admission	120 to 144	110 to 160
After three weeks	100 to 120	100 to 130
Blood pressure—		
Systolic	140	140
Diastolic	80	85
Urine—		
On 3 occasions contained small amount sugar, usually a trace		Trace of sugar in one specimen

Children such as these have movements which suggest chorea. The moist hands and the emotionalism of the two diseases are similar.

Among the observations made during the hospital course of nearly three months were records of the weight, various laboratory examinations, and basal metabolism.

The red blood counts varied from 4 to 5 million; white counts from 4 to 7 thousand.

The blood chemistry, serology, hemoglobin, were normal and identical as was the blood type.

Tuberculin tests were negative.

1931	R.				F.		
	WEIGHT			METABOLIC	METABOLIC	WEIGHT	
	LB.	KILO.		RATE	RATE	LB.	KILO.
Aug. 25	67½	30.61	1st reading	+68	+68	66¾	30.37
			2nd reading	+79	+68		
Sept. 9	70	31.75		+19	+30	68½	31.07
				+26	+28		
Sept. 28				+50	+84		
				+40	+71		
Oct. 3	74½	33.79				74½	33.79
Oct. 31	74¾	33.906		+48	+41	73	33.11
				+44	+46		
Nov. 12	71	33.205		+58	+38	74½	33.79
				+61	+48		

ESTIMATION OF THE INSENSIBLE PERSPIRATION[2, 3]

The insensible perspiration was estimated by weight observations at night under basal conditions. This corresponded well with the determinations of the metabolic rate. During the hospital stay the girls' weights and basal metabolic rate were closely related. At first R. pursued a course with lower rate and gained 8 pounds; then her weight fell off nearly 4 pounds and with it a higher metabolic rate developed. Her insensible perspiration likewise became greater.

November 15, 1931. Weight loss		
Ten-hour period	500 gm.	390 gm.
Per hour	50	39
On Benedict-Root straight line curve	2,030 cal.	1,700 cal. per day
Theoretical metabolism for age and weight	1,173 cal.	1,225 cal. per day
	+857	+475
Probable metabolism of	+ 73	+ 39 per cent

DUPLICATION OF DISEASES AS REPORTED IN THE LITERATURE

A brief mention of interesting instances in the medical literature might be made here. First it is well to recall that fraternal twins—originating from the fertilization of separate ova—may be of the same or of the different sex, but they do not resemble except as there is a tendency among all members of the family. True twinning is the

identical type, individuals of the same sex, who can with difficulty be told apart, unless environmental influences have produced differences.*

That these twins should show similar characteristics such as thinking alike and saying the same things, even when not in each other's hearing, fits in with the observations of Soukhanoff.[4] He found in six cases of similar mental disease among twins, that the mental processes in psychoses may be similar just as they are in health.

Palm and sole prints of newly born identical twins are identical. Wilder reported the similarity of the friction ridges on the hands and feet.

The similarity of congenital development may show such a rare solitary involvement as patency of the ductus arteriosus in both individuals (Shirley Smith).

Congenital absence of the upper incisor teeth as well as the presence of a goiter of moderate size in a set of twins were reported by Wilson and Wolfsohn.[5] These writers also recorded duplicate congenital ophthalmoplegia with feeblemindedness.

Twins with epilepsy were reported by Toledo.[6]

Mongolism is reported in one twin more commonly than in both. It has occurred in both twins in four instances.

Julius Hess[7] found Friedreich's ataxia in twin boys.

Identical twins had rickets, convulsions and tetany in an instance described by Abt.[8]

Retinoblastoma in homologous eyes occurred under the observation of Benedict.[9]

Identical twins had cerebellar tumors,[10] deaths occurring in childhood at an interval of two years.

As commonly as hyperplasia of the thyroid exists in adolescent girls, it is not to be doubted that a benign enlargement does similarly affect identical twins, probably not uncommonly. However, the occurrence of Graves' disease during childhood is not common as revealed by experience and by reference to the literature. Out of a total of 450 cases at the Royal Infirmary, Edinburgh, recently reported by Wallace,[11] only 4 cases occurred before the age of puberty. In 1,512 cases of exophthalmic goiter operated upon at The Mayo Clinic,[12] only five were in children under ten years of age. I have found no instance of exophthalmic goiter in twin children reported in the literature.

CONCLUSION

Graves' disease in both twins is rare, or not reported in children. What influence upon the goiter there may be at the completion of

*They spring from one ovum which ordinarily produces only one individual, for there is present in the ovary of the pregnant woman just one true corpus luteum. Identical twins, sometimes designated as homologous or duplicate, are embryologically known as monozygotic or univitelline twins. The incidence of this type in twinning is one to four.

adolescence, it is not possible to say, but the necessity for these girls to go to school and dwell in an environment not conducive to quiet may bring them soon to operation. It is the advice of several members of the hospital staff that thyroidectomy must soon be undertaken. The benefit which appeared in the first month may have been due to rest as well as to medication with iodine (Lugol's solution). The medicine was given during three periods of two weeks each. Its effect may have now worn out, a conclusion to which various observers have come in the treatment of this disease.

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ANTIQUES OF PEDIATRIC INTEREST*

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FROM the time of Aetius, in the sixth century, many notions and superstitions have persisted concerning the period of dentition. In my own childhood care was taken to burn my primary teeth after they had been extracted by the string attached to the door knob method; since, if they were thrown away and consumed by a dog, the secondary teeth would be similar in form to those of a hound.

As infants during dentition appear to derive relief from slight pressure on the gums, many objects have been given them to bite upon. These include the teeth of animals, including the colt's, boar's and wolf's, ivory, crystal, coral, silver and gold. Sylvius, 1682, considered the teeth of fierce, wild beasts most useful, since in addition to the pressing through of the teeth by the infant's biting "they send forth a volatile salt fume, very piercing, upon which accounts they help to cut the gums." Dionis, 1720, to the coral which cooled the inflamed gums and facilitated the cutting of teeth, added small bells to divert the attention of the child from its pains.

Teething necklace of viper's vertebrae, German, nineteenth century. Wolf's tooth for use during dentition, silver mounted, German, seventeenth century, 8 cm. in length.

Evanson and Maunsell, 1842, disagreed with Mason Good who believed that a gold teething ring produced ptyalism, and revived the ancient usage of allowing the child the use of a small wax candle, or occasionally a stick of liquirice root to bite upon, "But in truth a crust of bread answers all useful purposes."

Necklaces of amber, bones, roots, in fact beads of all descriptions were placed about the infant's neck, and the gums were anointed with ointments and lotions of all kinds during dentition.

George Armstrong, 1777, during teething "opened the body with a glyster, or a gentle dose of physic, if the case is not urgent, and afterward gave a puke; repeating it as occasion requires. If the child has a rash a blister is applied to the hind part of the head and kept running for some time."

John Cooke, 1769, thought the blister too irritating, so he applied leeches behind the ears, and anointed the hot gums with hare's brains or cold coral. If the pain was very bad he gave a drop or two of laudanum.

However, Rosen Von Rosenstein, 1776, concludes "that the brains of a hare, or the blood from the comb of a black cock, has no preference to other softening remedies; as also that a wolf's tooth is not preferable to either polished chrystal or any other hard substance."

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Critical Review

SURGICAL CONDITIONS IN CHILDHOOD

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PYLORIC Stenosis.—Donovan[1] reviews 119 cases operated upon by the Fredet-Rammstedt method. There were 6 deaths in the first 19 cases but only 1 in the next 100. The improvement in mortality is ascribed by the author to better preoperative preparation. Dehydration and undernourishment were corrected by blood transfusion, 20 c.c. per kilogram of body weight and one or more hypodermoclyses of 100 c.c. of glucose solution, 3 per cent. Ether was the anesthetic of choice. Water was given two hours after the operation and nourishment in the form of milk and barley water four hours later.

(Editor's note: The author's conclusions are in accord with the present general attitude that the operative treatment of pyloric stenosis is not an emergency, the best results being obtained if the operation is performed when the nutrition and fluid reserve have been built up to as nearly normal as possible. This often requires two or three days. When intravenous blood transfusion is difficult, intramuscular injections of 10 to 20 c.c. of whole blood are valuable and saline hypodermoclyses totaling 500 to 800 c.c. in each twenty-four hours may be required.)

Congenital Duodenal Obstruction.—Ladd[2] reviews the literature finding reports of only ten cases operated upon successfully. He reports ten additional cases three of which died.

The condition is due to maldevelopment from intrinsic or extrinsic causes. In the intrinsic type the epithelial lining of the duodenum undergoes proliferation, forming vacuoles which at one stage bridge the lumen with septa. If development is interrupted at this stage, the duodenum remains closed by a membranous obstruction. Under conditions of normal development the continuity of the lumen is reestablished. The extrinsic type is due in some instances to failure of complete intestinal rotation so that the entire small intestine remains attached to the posterior abdominal wall by a small area at the origin of the superior mesenteric artery. This permits a twisting of the mesentery and the formation of volvulus, the point of obstruction being in the fixed portion of the duodenum. The extrinsic type may also be due to a failure of descent of the cecum which becomes attached to the posterolateral abdominal wall just to the right of the duodenum and causes duodenal obstruction by pressure.

With each type the conditions may vary from stenosis to complete obstruction, and occasionally there is duodenal atresia. The symptoms are similar to those of pyloric stenosis except that the time of onset is usually much earlier, no pyloric tumor is palpable, and the vomitus usually contains bile. Surgery offers the only possibility of recovery, and the operations recommended are posterior gastrojejunostomy, pos-

terior duodenojejunostomy, untwisting of volvulus and freeing and derotation of the cecum. Ladd warns that the anastomosis in the small infant presents technical difficulties. The use of a distal loop clamp is unwise. Chromic catgut No. 000 should be used in the inner layer and should be reenforced with fine silk. The usual suture material is too coarse and may cause leakage. Operative findings, procedures, and results are well covered.

(Editor's note: This excellent presentation should be thoroughly studied by anyone attempting to treat such a case. Ladd's warnings are timely and should be carefully heeded. His experience, patience and delicacy in handling tissues are such that his success may be difficult to duplicate.)

Congenital Epulis.—This is a rare tumor occurring in the newborn as a rounded, somewhat pedunculated mass on the external side of the alveolar ridge. Moulonguet and De Lambert[3] review nine reported cases and present two additional ones. All cases on record have been in female infants. Occasionally the teeth which develop at the site of the mass are defective. The tumor is easily removed and no recurrences have been reported.

Undescended Testis.—Wangensteen[4] gives a review of the various theories which have been current regarding the physiology and development of the undescended testis. He states that there is failure of spermatogenesis but no interference with the development of secondary sex characteristics if the testis does not descend. The thermoregulating effect of the scrotum is necessary for spermatogenesis, and the testis will develop normally if placed in the scrotum before puberty. Up to the time of puberty few, if any, histologic changes can be noted. The author reports good results in 20 cases in which modification of the Torek operation was used. The basic principle of the operation is the fixation of the testis to the fascia of the thigh for a sufficient time to produce permanent lengthening of the cord and thus eliminate the possibility of later retraction.

The usual careful dissection and lengthening of the cord are done, the scrotal pouch is formed, the testis is brought out through the side of the scrotum, anchored to the fascia of the thigh and the edges of the scrotal and thigh incisions united about the testis. For one week the thigh is kept in a position of flexion after which full motion is encouraged. The union is severed after four months and the testis remains in the normal position.

Walters[5] reports 100 cases of undescended testis operated upon during the past five years by the method of Torek and Meyer, with no deaths and uniformly good results. He leaves the testis attached to the fascia of the thigh only four weeks. The cord can always be lengthened sufficiently, and the method is applicable even to intraabdominal testes.

(Editor's note: The Torek operation, although not new, has never received as wide recognition or become as popular as it deserves. The fault common to most of the usual operative procedures is the late retraction of the testis to the upper part of the scrotum or even to the external ring with consequent failure of spermatogenesis. This of course, nullifies the effect of the operation. The objection raised to the Torek operation is that it requires two or more stages, but the percentage of good results is so much greater than that obtained by other methods that the objection does not seem valid. While the operation

may be done at any convenient time before puberty, it is our observation that the later years of childhood are preferable to the earlier.)

Skull Fractures.—Ireland[6] reports a series of eighty cases in children up to the age of twelve years with two deaths. One year after injury only nine children showed sequelae. The most constant symptom was vomiting. Unconsciousness was present in 40 per cent. High temperature and pulse are not of particular significance. Fixed and dilated pupils indicate a serious injury with unfavorable prognosis. Lumbar puncture is indicated only when signs of increased intracranial pressure are present, and should be done slowly and carefully. Decompression is indicated if the symptoms are not relieved by lumbar puncture. Fractures which are only slightly depressed and causing no symptoms do not require operation even though they may overlie the sinuses.

(Editor's note: The policy of limiting operative procedures strictly to those cases which present definite indications for surgery is responsible for a marked decrease in the mortality rate of skull fracture in children. Early treatment of shock with a minimum amount of disturbance to the child, dehydration when there are symptoms of concussion, accurate recording of pulse and blood pressure at short intervals, and emergency surgery only for the control of hemorrhage, are the essentials of accepted treatment, and departure from them is courting danger. Lumbar puncture is frequently unnecessary when dehydration can be effected by the use of hypertonic solutions by rectum or vein.)

Spina Bifida.—Coughlin[7] reports a series of twelve cases in which closure was performed between the ages of sixteen hours and seven months. In no case was there a succeeding hydrocephalus. There were three deaths. No improvement was noted in sphincter paralysis. Of six cases showing paralysis of the extremities one showed slight improvement, and one was worse following operation. Eight cases were draining spinal fluid. The operation is performed upon an angulated table with the head down, which position is maintained for at least six days afterward. The buttocks are isolated from the operative field by rubber dam glued to the skin. The base of the sac is carefully isolated through an ample transverse incision. All nerve elements are carefully preserved and are restored as nearly as possible to their normal locations. The defect is covered with a flap of the lumbar fascia. Reconstruction of the bony canal is contraindicated as it is unnecessary and increases shock.

Penfield and Cone[8] in a study of the literature on this subject find a general operative mortality of at least 50 per cent. They report a series of 31 cases and propose a new operative procedure. Twenty-one cases were operated upon with two deaths, no development of true hydrocephalus or mental defect, and only four noticeably large heads. In the ten remaining cases operation was either refused or else contraindicated by the severity of the associated conditions. One of the operative deaths occurred in a case of anterior cranium bifidum with the meningocele presenting through the palate and the other in a case of multiple craniospinal defects and a large ventral hernia. In both of these cases operation was possibly ill advised.

The feature of the authors' operation is the preservation of the sac which their extensive studies have shown to have definite powers of absorption of cerebrospinal fluid. The postoperative care is of great

importance. The head is constantly kept down during and after operation until healing is complete. Then it is raised and kept elevated at all times, the horizontal position being never permitted even temporarily until there is no further evidence of increased intracranial pressure. With the cooperation of a pediatrician the fluid intake is reduced to a minimum and solid food begun as soon as possible. The period of postoperative observation has varied from one to twenty-eight months with the exception of one case which has been followed for eight years. Paralysis existing before operation is not changed, and signs of intracranial pressure are frequently increased temporarily, eventually, however, disappearing. Fluid and an expansile impulse can often be palpated in the sac for several years.

(Editor's note: These reports are of special interest in that hydrocephalus has not followed early operation. The function of the sac in the absorption of cerebrospinal fluid is a valuable new concept. The necessity of adherence to a strict and prolonged postoperative regimen may tend to limit the benefits of this treatment to institutional cases and to those in the upper economic strata, but the authors are to be congratulated upon bringing encouragement into a field in which results have been so often unsatisfactory.)

Osteomyelitis of the Jaws.—Wilensky[9] gives a comprehensive discussion of this serious and often fatal condition. It occurs usually between the ages of two and ten weeks. The initial symptoms may be mild but are often severe, consisting of high fever, convulsions and vomiting. These are followed by swelling of the cheek, edema of the lower eyelid, occasional exophthalmos, and localization usually below the inner canthus with abscess and fistula formation. In some cases there is swelling of the alveolus with fistulous tracts discharging into the mouth or nose. Sequestra and unerupted teeth may be discharged until the major portion of the superior maxilla is destroyed. The resulting deformity in case of survival is extreme.

In the differential diagnosis, erysipelas, syphilis and tuberculosis are to be considered, and the eye symptoms may cause confusion with dacroeystitis and ophthalmia neonatorum.

The treatment consists of careful cleansing of the mouth, conservative treatment of the nasal and orbital conditions, incision and drainage of fluctuant areas through the mouth and nose wherever possible and systemic treatment to develop resistance.

Empyema.—In the portion of his article which refers to children Bettman[10] states that the method of repeated aspiration is still occasionally used but is no simpler than the introduction of a catheter through a trocar. Local anesthesia is employed and a technic used which prevents the entrance of air. Sufficient pus is removed to relieve respiratory embarassment, a maximum of 250 c.c., and aspirations are repeated every three hours followed each time by injection of Dakin's solution. Blow bottles are used to hasten expansion of the lung, the end of the catheter being open at such times to permit escape of the air.

Obliteration of the cavity under this treatment usually requires about six weeks.

In the presence of bronchial fistula, saline instead of Dakin's solution is used, and the end of the catheter is submerged in water.

The closed method of drainage is as adequate in bilateral as in unilateral empyema.

If pneumonia is present aspiration should be done more frequently and in smaller amounts, but whenever possible treatment should be postponed until pneumonia has subsided.

Encapsulated or interlobar empyema may require rib resection.

(Editor's note: The theory and application of closed drainage are still controversial subjects, although it is undoubtedly the procedure of choice in infants. The advocates of the open method point out many inconsistencies in the statements of their opponents and are able to produce equally good statistics. The reasonable position would appear to be that the type of infection and the condition of the patient should determine the type of drainage.)

Intussusception.—Shelley[11] gives a concise review of the methods of treatment of the more severe types of intussusception. Reduction may at times be accomplished by pressure enemas or air insufflation, but this should always be followed by abdominal exploration for confirmation. During operative reduction the intestines must be protected against drying and mechanical irritation. Edema may be reduced by firm continuous pressure. No traction should be made upon the involved bowel, reduction being effected by gentle, continuous, backward pressure upon the apex of the intussusceptum. Various treatments mentioned for the irreducible type include incision of the neck of the intussusception with subsequent repair of the opening, enterostomy above the lesion, lateral anastomosis about the lesion, and resection. If resection is necessary, the "double-barrel" type is the procedure of choice, as the time and manipulation necessary for anastomosis usually result fatally. Where there is perforation into the peritoneal cavity, the affected loop may be brought through the incision and a Mikulicz type of drainage done.

Ether is the anesthetic advised. Preoperative and postoperative care should be particularly directed toward combating dehydration.

The appendix should not be removed even though its inclusion in the intussusception has given it the appearance of acute inflammation.

The necessity of a general anesthetic is well shown in a case of a seven months' infant in which operative reduction was being demonstrated by Andrews.[12] The operation was begun under local anesthesia but it was necessary to supplement this with ether and ethylene before satisfactory quiet and relaxation could be obtained.

An analysis was made by Close[13] of 363 cases treated in Guy's Hospital during the years 1904 to 1927 inclusive. The ages varied from eight weeks to twelve years, and the author refers to a report of a case occurring in an infant two days old. There was a family history of intussusception in 146 cases. There were 14 instances of recurrence; in two of which the condition recurred three times. The mortality was 31 per cent most of the deaths being in patients under the age of one year. Resection was done in 17 cases with 100 per cent mortality, and in one case of gangrenous intussusception without resection the patient survived. In this case spinal anesthesia was used and an anastomosis done around the lesion. The mortality has been reduced from 40 to 20 per cent during the period under survey due, in the author's opinion, to improved anesthesia.

(Editor's note: Shelley's warning against unnecessary manipulation and appendectomy is of vital importance and should be heeded. The use of local anesthesia is of doubtful value, as infants tolerate ether or

ethylene very well. Struggling increases operative and manipulative trauma and expends energy which should be conserved for postoperative recovery. Spinal anesthesia gives excellent relaxation and collapsed intestines but should be used with caution, as sufficient statistics are not yet available regarding dosage in infants.)

Pyelitis.—Lack of knowledge on the part of the great bulk of the medical profession of modern advances in urologic diagnosis and treatment is considered by Sprenger[14] as responsible for an unnecessarily high mortality and morbidity from urinary conditions in children. With the development of infant cystoscopes and ureteral catheters all of the diagnostic procedures possible in adults can now be used in infants and children. General anesthesia is usually desirable particularly in the male, and with the exercise of care and gentleness trauma is unnecessary. Reactions following ureteral catheterization are mild or absent.

Congenital defects are a frequent cause of urinary abnormalities, and remedial measures will often clear up persistent pyuria and fever. The diagnosis of pyelitis is frequently incorrect, as careful examination has shown the lesion to be usually in the kidney and rarely in the renal pelvis.

Pathologic conditions discovered include hydronephrosis, ureteral stricture, suppurative nephritis, renal tuberculosis, ureteral diverticulum, and reduplication. Urinary antiseptics have not been as effective in inhibiting bacterial growth as copious fluid intake combined with alternate alkalinization and acidification of the urine.

The author reports cystoscopy and pyelography in a male infant of three weeks.

Clark[15] reports fifty cases of bacilluria treated by a strictly regulated ketogenic diet supplemented in some cases by oral administration of ammonium nitrate and bladder lavage with dilute acetic acid. Certain bacterial strains were found to be resistant, and careful cultural study was an aid in prognosis. Results are listed as satisfactory in 66 per cent of the series, and of those listed as unsatisfactory all but two showed definite clinical improvement. The two essential factors are ketonuria and a definitely low urinary P_H. Investigations by Helmholz[16] have shown that the urine is not definitely bactericidal or bacteriostatic unless the urinary P_H ranges from about 5.0 to 5.6, and Clark considers that it must not be above 5.2. The results listed as unsatisfactory were attributed to absence of ketones, poor renal function, insufficiently low P_H, poor dietary cooperation, or too brief period of treatment.

(Editor's note: Unquestionably many cases of persistent pyuria are not caused by pyelitis, and the use of instruments and methods particularly adapted to infants and children permits much more accurate diagnosis and effective treatment than has formerly been possible.

When these diagnostic aids are available any pyuria which resists the usual forms of treatment, especially when associated with fever, should be subjected to a careful study.

The ketogenic treatment presents certain technical difficulties when applied to everyday practice, on account of the necessity of strict dietary supervision, close cooperation, and the maintenance of so narrow a zone of urinary variation. However, it is in keeping with the present trend of using physiologic rather than empiric methods wherever pos-

sible, and may become of distinct value in institutions where the necessary facilities are available.)

Thymus.—Routine preoperative x-ray pictures of children to determine the condition of the thymus are unnecessary. Brittain[17] enumerates certain symptoms which may indicate thymic enlargement, and advocates diagnostic and therapeutic use of the x-ray only if one or more of them are persistent. They are intermittent cyanosis, convulsions of obscure origin, dysphagia, noisy nasal breathing, breath holding, brassy cough, spasmodic inspiratory stridor, and dyspnea. Lateral views are advisable in all suspected cases, as there may be no increase in breadth of the shadow. The author warns against indiscriminate x-ray treatments.

(Editor's note: In the much debated field of thymic enlargement, persistence, and death, the trend of opinion seems to be to disregard x-ray evidence when it is not supported by clinical findings. The shadow may show enlargement when there are no symptoms, or there may be definite symptoms which disappear promptly upon treatment, without demonstrable enlargement. Until we are in possession of more accurate information upon this condition the rational procedure would seem to be to use the x-ray as a therapeutic agent and not place too much reliance upon it as diagnostic aid.)

Umbilical hernia.—In an analysis of 349 cases Miller[18] finds that 17 per cent were strangulated. The mortality in these was 30 per cent, while in the simple cases it was 2.4 per cent. The great majority of the cases were in adults. The author states that operation of election for simple hernia is safer than the scientifically calculated expectancy of life in these cases and therefore that operation should always be urged in preference to the use of mechanical supports. In the discussion it was brought out that in children the use of a button strapped down upon the navel separates the muscles and interferes with the normal process of closure. A plain adhesive strip is all that is necessary. A hernia persisting until the age of six years should be repaired. Strangulated cases require careful technic and often should be operated upon in two stages, the first being simply a cutting of the ring under local anesthesia to permit the circulation to be reestablished.

(Editor's note: It is doubtful whether the use of a button or similar device has ever produced a cure which could not have been effected by simple strapping. It has been our observation that when the ring is large enough at the age of three years to permit a marked extrusion spontaneous closure does not occur. Miller's statistics upon the mortality from this condition in adults should be a potent argument for an operation of election in childhood.)

Hemangioma.—The use of radium and roentgen rays in 40 cases is analyzed by Roesler.[19] Twenty-five were under two years of age. The tumors which are present at birth or in infancy show a definite tendency to grow. In eleven cases treatment caused the tumor to disappear and the skin was normal; in 20 cases the tumor practically disappeared but the skin showed slight atrophy, induration, discoloration or depigmentation. In 10 deep-seated tumors the results were in general good. Progress often continues for some time after treatment, and final judgment should not be passed for at least six months. Many cases require several treatments, but great care should be exercised because of

the possibility of disturbances of tissue growth with scar contraction. The results are definitely better in children of two years and under. Ulceration is not a contraindication.

(Editor's note: Treatment by the water-cooled ultraviolet light and by freezing was not under discussion in this article. Such treatment in suitable cases produces excellent results and may be used when the special training so necessary for the safe employment of the radium or roentgen treatment is not available.)

Burns.—A study of the tannic acid treatment of burns in 310 cases made by Glover[20] and a similar study by Strauss[21] of 352 cases show clearly the superiority of this method over former ones. In these series between 40 per cent and 50 per cent were in children. The mortality during the first twenty-four hours, the period of initial shock, was not changed, but that of the succeeding periods was markedly less than under previous methods. Shock should be treated first, the burned area cleaned, and 5 per cent tannic acid sprayed directly on the burn at frequent intervals for twenty-four hours or until a hard dry coagulum is formed. This is followed by open air treatment under a cradle with the temperature maintained at approximately 85° F. Fluids should be forced to combat dehydration, but Glover warns that judgment must be used to avoid edema during the first three days before the kidneys have recovered their full excretory function. He considers that the amount frequently recommended of 100 c.c. per kilogram of body weight a day is too high. The tanned coagulum is removed by moist compresses of Dakin's solution, one observer using it only if there is evident infection and the other on the seventh day. Early skin grafting of granulating areas is strongly urged as the tannic acid treatment does not promote epithelialization, and secondary anemia is liable to develop.

Vomiting and convulsions may occur in the toxic stage and require blood transfusion. Exsanguination transfusion is of great value and Strauss emphasizes the necessity of passing the needle through the saphenous vein into the femoral in infants and small children. The jugular vein is a second choice.

Comparative mortality statistics should be accepted with some reservation because of the great number of variable factors involved.

(Editor's note: The seriousness of burns in children is shown in one of these series by the fact that the mortality under ten years of age was 22½ per cent as compared with 16½ per cent for the entire series. The skin area of a young child is greater in proportion to the body weight and blood volume than in adults, which may be a factor, although others no doubt enter into the picture. The articles are excellent in their subject matter and attention to details.)

Hirschsprung's Disease.—Rankin and Learmouth[22] report the results of eight cases treated at The Mayo Clinic by resection of the lumbar sympathetic trunk or the presacral and inferior mesenteric nerves. These nerves are the inhibitory or "filling" nerves of the colon and are the logical point of attack if the neurogenic theory of this disease is accepted. The newer technic spares the vasomotor nerves to the lower extremities. Five of the cases were in children of twelve years or less, and the results were all good from two to twelve months after operation.

(Editor's note: As the authors note, there may be other causes for this rather obscure condition but their treatment is apparently effective in the neurogenic type. Their future reports will be awaited with interest as to final results. The body possesses marvelous powers of readjustment, and it is within the realm of possibility that the unknown cause of this condition is still present and will stimulate the formation of new nerve pathways to take the place of those which are severed.)

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SECOND ANNUAL MEETING OF THE AMERICAN ACADEMY OF PEDIATRICS

FRIDAY AFTERNOON SESSION
May 13, 1932

Round Table Conference on Rheumatic Infections

Leader: Dr. John L. Morse; Assistants: Dr. Hugh McCulloch and Dr. Albert D. Kaiser

Rheumatic Infections

John Lovett Morse, M.D.

RHEUMATISM is now believed to be a disease in which the whole circulatory system is involved. Its symptomatology necessarily varies according to which part of the circulatory system is most involved at the given time; hence, articular rheumatism, cardiac disease, chorea.

There appears to be a close association between streptococci and the rheumatic infections. It is safe to say that, although various claims have been made as to the discovery of a specific strain, no especial type, whether indifferent or hemolytic, has been proved to be the etiologic agent. While some observers have found streptococci in the blood, most have not. The same is true of the joints. The evidence is, therefore, against a blood-stream infection. The pathologic changes suggest that the disease is a manifestation of hypersensitiveness to the products of streptococci. It is believed that this hypersensitiveness develops as the result of repeated streptococcal infections, usually in the upper respiratory tract or fauces, or of chronic streptococcal foci of infection, most often located in the tonsils, sometimes about the teeth, occasionally elsewhere. Then, when an infection with streptococci occurs in the upper respiratory tract, as is claimed to be almost always the case, the hypersensitive tissues overreact and the symptoms of rheumatism develop. This usually happens after an interval of two or three weeks.

In a very general way, as the result of the action of the toxic products of streptococci on the hypersensitive tissues there is swelling, cellular infiltration and necrosis of the mesenchymal tissues. The exudative and proliferative tissue reactions are all out of proportion to the tissue necrosis. These changes are not, however, specific for rheumatic fever, as they have been seen in other streptococcal diseases. Aschoff's bodies, which are formed as the result of the action of histiocytes and fibrocytes, develop in from three to six weeks. The changes in the mesenchymal tissues gradually disappear, and the Aschoff bodies change to connective tissue nodules, that is, if there are no further streptococcus infections or foci of infection present. If there are, recovery does not take place and all stages of

the pathologic process may be found at the same time. It seems evident that there is nothing in the pathologic process itself to justify the assumption that recovery is not possible.

It has been known for many years that the articular symptoms of rheumatism are milder in children than in adults, that children are more likely than adults to develop heart disease in rehumatism, and that they may develop heart disease without any of the usual evidences of adult rheumatism. These are not new discoveries. I was taught this when I was a medical student. We were also taught that "growing pains" were often really rheumatic in origin. Now we must not forget that a child may have pains which are not due to rheumatism.

It is the fashion now to call many things rheumatic, which a few years ago were not supposed to be, while forty years or more ago they were. Some of these are abdominal pain, either acute or chronic, and acute pleurisy. There is danger of forgetting appendicitis and tuberculosis if too much is thought of rheumatism. Nosebleed is more often due to picking the nose or to localized dilatation of blood vessels than to rheumatism or heart disease. There are many other causes for malnutrition and poor health besides rheumatism. Continued fever may be due to tuberculosis. That is, there are other causes for the symptoms which rheumatism may produce besides rheumatism. The tendency at present is to attribute too much etiologic importance to rheumatism and too little to the other possible causes for them.

An important step forward has been taken, however, in the realization that rheumatism is a chronic disease or at any rate one with frequent slight recurrences, and that it should always be thought of when children are below par, run a slight fever, have a continuously elevated pulse rate or leucocytosis, even if there are none of the more acute evidences of rheumatism present.

It has been known for many years that children were very likely to develop heart trouble after tonsillitis and rheumatism. It was also taught that heart disease might be the earliest or only symptom of rheumatism. It was well known that all parts of the heart were involved. Sturges wrote a book about the acute carditis of childhood in 1895. The pathologic changes and the reason for them were not, however, as well understood. That is, it was not realized that the disease process was vascular and that, therefore, all parts of the heart must be involved. It is not necessary to speak of the myocardial changes. It is important, however, to remember in relation to the symptomatology that the pathologic process starts in the vascular region of the valve ring and extends toward the edge of the leaflets. Transitory murmurs are better explained, therefore, by the diffuse infiltration of the valves and the resulting stiffness than by dilatation of the ring. They resume their function with the disappearance of the edema and the murmur ceases. Injuries to the valves at the line of contact may result mechanically. Permanent dysfunction may result from scarring and contraction. The lesions in the myocardium may, of course, be increased as the result of the chronic recurring nature of the process, and the cardiac condition grow progressively worse. As long as the pathologic process is active it is proper to speak of it as rheumatic heart disease. If the active process has stopped, then it is not rheumatic heart disease. The cardiac condition is then purely a mechanical one, the result of rheumatic heart disease. It is claimed by some that the cardiac lesions of rheumatism are never healed. There is nothing about them which prohibits healing. Tuberculous lesions heal, why not rheumatic? The focus of infection can be removed and the upper respiratory infections stopped. Rheumatism becomes much less common after the age of twelve or fifteen years, presumably because the condition of hypersensitiveness has been overcome. There is then no further cause for the continua-

tion of the pathologic process in the heart, and no reason why it should not stop and heal. Clinically, at any rate, the cardiac inflammation does stop and recovery occurs.

If a child is believed to have rheumatism and the present conception as to the pathology of the disease is accepted, it is necessary to assume that the heart is always involved to a greater or less extent. As long as the infection lasts or recurs it is possible, or even probable, that it may be seriously damaged. The persistence of pains, choreic manifestations and subcutaneous nodules is, of course, certain proof of the activity of the disease. Persistent fever, even if of slight degree, and leucocytosis, provided no other cause is found for them, also almost prove that activity persists. A persistently rapid pulse rate points strongly to involvement of the heart. Does it prove it? Prolongation of the auriculoventricular conduction time by electrocardiogram is important evidence of involvement of the heart. Of how much value as negative evidence is the absence of such prolongation? Of very little, I fear. Of how much value is an increased sedimentation rate of the red blood cells? Of none to the general practitioner. Finally, and of the greatest importance, how much attention shall be paid to these findings in deciding on the treatment to be followed and what shall this treatment be?

The most important element in the treatment of all diseases is the preventive. It would be ideal to have all children well-to-do, to have them live in warm climates, and to protect them against all upper respiratory infections. It would be fine to send them to a warmer climate in the winter if they showed any evidences of rheumatic infection. Unfortunately we can only aspire to such ideal conditions.

It would seem as if the removal of all foci of infection would markedly influence the incidence of rheumatism. Nevertheless, does removal of the tonsils and adenoids diminish the incidence of rheumatism? Does the removal of diseased tonsils diminish the number of recurrences or favorably influence the course of cardiac disease? Should the tonsils be removed in the midst of an attack of rheumatism or not? Does the removal of the tonsils do harm? None of these questions, to my mind at least, have been satisfactorily answered. However, it seems reasonable to keep on removing tonsils if they appear to be diseased.

It is now believed that rheumatism is the result of hypersensitiveness to the products of streptococci. It is well known that the incidence of rheumatic manifestations diminishes very materially after the age of twelve to fifteen years. It is evident that nature in the course of years in some way, possibly by autodesensitization, overcomes this hypersensitiveness, and hyposensitiveness develops in its place. It would seem reasonable to attempt to induce hyposensitiveness by artificial desensitization. Swift and his coworkers have attempted to do this by intravenous vaccination with hemolytic streptococcal vaccine. They found that the incidence of recurrences was less in the treated children than in the controls. They did not feel justified, however, in drawing any positive conclusions as to the results of this treatment, but recommended that at present it should only be used in controlled investigations.

Another reasonable form of treatment during recurrences would seem to be antistreptococcic serum. Several types of this serum have been used. At first striking results were claimed, as is the case with all new methods of treatment. These results have not been corroborated by others. Severe reactions are not infrequent. At present, therefore, it seems fair to conclude that this method of treatment is not justified by the results thus far obtained.

The salicylates have been used for many years in the treatment of acute rheumatic manifestations. Opinions still differ as to whether they have some curative action or simply relieve symptoms. Opinions still differ also as to whether they are of value or not in preventing recurrences or in preventing involvement

of the heart when other rheumatic symptoms are present. The salicylates, their action and value offer a fertile field for discussion.

It is generally admitted, I think, that rest in bed is an essential part of the treatment of rheumatism. It is also generally admitted, I believe, that rest in bed is the most important thing in the treatment of rheumatic heart disease. How long should this rest be? How shall we know when to get children out of bed? Should they stay in bed as long as the temperature is elevated, as long as there is a leucocytosis, or as long as the pulse is rapid? Should they stay longer than this? If they should, how much longer? Will rest in bed prevent clinical involvement of the heart in rheumatism? How long should children with rheumatic infection without clinical involvement of the heart be kept in bed? What are the symptoms of beginning involvement of the heart?

Almost everyone agrees, I think, that digitalis should be used when compensation fails in chronic valvular disease of the heart. Some claim, however, that there is no such thing as chronic valvular disease when it is rheumatic in origin. According to them it is always active rheumatic heart disease. However that may be, what is now called congestive failure does occur in hearts in which the symptoms are due to mechanical conditions without any manifestations of active rheumatic infection. Granting that digitalis usually does good under these circumstances, does it do good and should it be used when manifestations of active rheumatic infection are also present? Should it be used when the cardiac condition is new and there are no old valvular lesions? Will it do good or harm at this time? This is also a fertile field for discussion.

There are many other questions in connection with the etiology, symptomatology, prognosis and treatment of rheumatism which are well worthy of discussion and about which there must necessarily be much difference of opinion. It is possible that time may show that all our present conceptions are erroneous. Stranger things have happened in medicine than this. All that we can do, however, is to keep an open mind, avoid both undue credulity and skepticism, and endeavor to reason and act sanely and safely.

DISCUSSION

The question of diagnosis of rheumatic infections was discussed freely. It was felt that in the usual child with acute rheumatic infection this diagnosis was easily made but that in many children, especially younger ones, it was often difficult to recognize. This difficulty centers chiefly about the question of ill-defined pains in the extremities which are usually called growing pains. It was agreed that there is no reason why growth in itself should cause pain in the body and that this term is a misnomer. Dr. Kaiser pointed out that a better term than growing pains in reference to rheumatic fever would be joint pains. This would be taken to mean actual pain in and about the structures of the joints associated with objective signs. Dr. Emerson stated that frequently children had pain in the extremities due to excessive activity, and these pains were difficult to differentiate at times from rheumatic fever. Dr. McCulloch pointed out that this type of pain would best be described as fatigue pain; that it is usually localized to the lower extremities and they are not associated with objective changes in the joints and are frequently present at the end of the day and during the early evening. These pains occur frequently in suspected rheumatic children because they usually are underweight and have a poor response to exercise. It was also mentioned that flat feet may cause pain in the extremities of children and that this should be kept in mind in diagnosing a child with suspected rheumatism.

In response to a question as to the etiology of rheumatism several important factors were emphasized. It was pointed out particularly that the disease seems

more concentrated in urban localities though it occurs occasionally in individuals living in the country. The effect of bad housing, overcrowding, insufficient food and lack of opportunity for outdoor play are most important factors in the increased incidence in urban life. It was also pointed out that sunlight through its ultraviolet activity may have some influence in the incidence of the disease especially through its effect on acute infections. Dr. Kaiser pointed out that there seemed large localities where rheumatic fever is entirely absent among the natives. He recalled the recent observations which have been made in Porto Rico where it is very unusual to find an individual with rheumatic heart disease or rheumatic fever. However, when Porto Ricans come to New York City to live, the incidence of the disease is fairly high among them. He also recited the recent experiments in New York when rheumatic children were taken to Porto Rico for a long period of time. While there, they remained free of the disease and improved a great deal. Some of these children on returning to their homes in New York again developed signs of the disease.

In discussing the etiology of rheumatic fever, the very important question of the part played by the tonsils was discussed. Dr. Mitchell stated that in his opinion this question cannot be definitely decided at the present time. That in his recent work trying to clear up some of the relations that exist between infected tonsils and systemic disease in children he has been impressed with the difficulty in determining the exact relations that exist. He emphasized the well-known fact that the tonsils are a part of the lymphatic tissue of the nasopharynx and that the portal of entry frequently may be through this tissue in other areas than the tonsils. This question is most important to decide in order to have some opinion as to the value of tonsillectomy in these children. If the tonsils were clearly infected and showed the usual signs of disease, then they should be removed, but there are many children with signs of systemic disease in whom the tonsils may not be so clearly involved and so closely related to the disease. Further careful studies seem to be necessary to settle this point. Oftentimes it is an individual question with the child, and the judgment of the physician who is familiar with the child and the situation must be relied upon.

Dr. Emerson in reply to the question: when should a child with acute rheumatic disease be permitted to get up, stated that not only absence of fever should determine this but a pulse rate of more than 100 contraindicated getting out of bed; a leucocytosis of more than 10,000 likewise showed signs of activity. When the fever was absent, pulse less than 100 and leucocytosis under 10,000, the child could safely get up. Considerable discussion followed by various participants as to what constituted a normal temperature. Dr. Mitchell stated that many well children regularly had a rectal temperature of 100° F. He advocated taking the temperature of well patients so any supposed elevation in temperature could be compared with the normal for that child.

In answer to the question: are x-ray studies of the heart of any value in determining the condition of the heart in rheumatic disease, several opinions were expressed. Dr. Emerson and Dr. Mitchell felt that the x-ray was an aid in evaluating the condition of the heart. Physical examination might give the same information, but in some instances incorrect conclusions would be arrived at without the x-ray examination.

Dr. Morse asked: What should be done with foci of infection as a factor in rheumatic disease? Dr. Kaiser then again discussed the part the tonsil plays in the rheumatic disease. Where the tonsils show definite evidence of infection or if a history of repeated attacks of tonsillitis is given, the tonsils should be removed even though statistical evidence shows no great value in this procedure. In indi-

vidual cases recurrent throat infections may be avoided and thus influence favorably the rheumatic infection. The possibility of sinus infection as a focus was admitted. The general opinion was that sinus infection rarely needs to be treated for it usually disappears during the summer weather. Dr. Morse was opposed to any sinus treatment in children except in exceptional cases.

Dr. Wilkes discussed the value of careful health examinations in finding children with rheumatic heart disease. He lamented the fact that in some communities such examinations were not carefully made and that removal of the tonsils would be advocated at the direction of a nurse rather than upon the advice of a competent physician.

Dr. Morse closed the discussion by summarizing the known facts in this disease and stressed the importance of treating for a long period children who are suspected of having rheumatic infection even if the signs and symptoms are not definite. A prolonged rest period as long as three months is necessary where an initial cardiac infection is suspected. With our present knowledge of the disease he feels this is the most important procedure. The unknown factors were reviewed, especially those that have to do with the etiology of the disease and the constitutional factors in individuals susceptible to rheumatic disease.

Academy News

The following members have been appointed state chairmen in their respective states.

Maryland: Dr. Fred Bruce Smith, 11 E. Chase Street, Baltimore.
Ohio: Dr. Charles W. Burhans, 2103 Adelbert Road, Cleveland.

Dr. Alfred F. Hess has been elected a member of the Deutsche Academie der Naturforschung.

Dr. S. McClintock Hamill is now convalescing satisfactorily after a severe illness, with surgical intervention.

News and Notes

Dr. Ralph M. Tyson has been appointed Professor of Pediatrics at Temple University, Philadelphia.

On June 23, 1932, the **Section on Pediatrics of the West Virginia Medical School** held its first meeting in Parkersburg, West Virginia. Dr. George M. Lyon was elected Chairman and Dr. M. F. Petersen, Secretary. The meeting was well attended and the establishment of the Section on Pediatrics was enthusiastically approved. Dr. A. Graeme Mitchell of Cincinnati was present as the honor guest. This Section was organized only last year.

The **North Pacific Pediatric Society** held its midsummer meeting on June 25 at Tacoma, Washington. Dr. Temple Fay of Philadelphia was the invited guest.

The **Oklahoma Pediatric Society** held its annual meeting in Tulsa May 24. Dr. Frank C. Neff of Kansas City was the out-of-state speaker. The following officers were elected for the coming year: Dr. Clark H. Hall, Oklahoma City, president; Dr. George H. Garrison, Oklahoma City, vice-president; Dr. M. J. Searle, Tulsa, secretary-treasurer. The next meeting will be in Oklahoma City in May, 1932.

The **Oklahoma State Follow-Up Committee** of the White House Conference on Child Health and Protection was held at the Southwestern State Teachers College, Weatherford, Oklahoma on June 28, 1932. The meeting was attended by about 1,000 people and there seemed to be considerable interest in the proceedings.

The **Second Conference of the International Association for Preventive Pediatrics** will be held in Geneva on September 28 and 29, 1932. Dr. Taillens of Lausanne is president. The subjects placed on the agenda are: (1) The Prophylaxis and the Treatment of Syphilis in Children Born of Proved Syphilitic Parents. Reporters: Prof. Lesné of Paris, Dr. Nabarro of London, Dr. Gösta Ahman of Gottenburg. (2) The Prophylaxis and Treatment of Neurosis in Children. Reporters: Dr. Heuyer of Paris and Prof. Hamburger of Vienna.

Book Reviews

Nutrition Service in the Field. Child Health Centers: A Survey. Reports of the subcommittees on Nutrition and Child Health Centers. The White House Conference on Child Health and Protection. The Century Co., New York, 1932. Pp. 196.

Nutrition Service in the Field

According to the most conservative estimates, six million of our forty-five million children in the United States are malnourished or one in every six or seven. As every physician recognizes the importance of good nutrition and the medical profession has done so much to make good nutrition possible for children this statement is very challenging. Why are there so many malnourished children in all economic levels?

In an endeavor to analyze this situation the Section on Medical Service requested a subcommittee to find out what nutrition service exists, how it operates in health programs and to make recommendations for making optimum nutrition more possible for children.

This publication gives the findings of the Committee and describes the various types of nutrition work being done throughout the country. It indicates there are relatively few trained people giving full-time service to nutrition education and that many groups in positions to disseminate facts are not doing so.

In view of the apparent need for a broad educational program the Committee recommends:

1. That nutrition work be organized as a basic part of every district, county or community program for child health and protection.

2. That nutrition programs in public health and welfare agencies and educational systems be organized under accepted medical direction.

3. That one or more qualified nutritionists, either on full time or on part time, be connected with every child health program.

Close correlation between medical and nutrition service is evident in these quotations:

"Nutrition work has two aspects, one corrective in character and the other preventive. Both must receive adequate attention in any thoroughly constructive plan for child health."

"While the remedial aspect was the first to claim attention we are slowly learning that a steady day-by-day building for good nutrition is still more important. This procedure has its roots deep in the educational and preventive measures."

Various organizations have aimed to incorporate these two aspects of service in their child health programs. The appendix of this report discusses in detail the development and present nutrition service of twenty-two agencies doing outstanding work along these lines. Physicians will find this one of the most constructive parts of the report.

MRS. ANNA DEPLANTER BOWES.

Child Health Centers:

More than a million children are being given formal health examinations and health advice annually in 1,511 health centers in cities of the United States. A description of these centers and a very careful analysis of their work is presented in this volume.

This information, assembled now for the first time, comes at a moment when it should be of the greatest possible value to physicians, hospitals, public health agencies and social workers, all of whom are deeply concerned with the physical and mental hazards which the children of the country are facing in the present economic crisis.

Here are opportunities for examination—facilities and health supervision within the reach of destitute families. Here is a bulwark against the imminent flood of malnutrition, tuberculosis and nervous disasters with which we are threatened by today's economic chaos. Every community where there are children growing up without medical supervision, this report claims, needs an agency or organized plan through which parents with little or no funds can bring their young children for examination and advice. It should not be necessary for these citizens to depend upon the charity of the individual physician nor should the busy practitioner with his living to make, be obliged to devote an undue amount of his time to the routine examination of and advice to patients without remuneration.

Only a small proportion of the children examined through health centers have been under regular medical supervision before.

This type of preventive care, scarcely used prior to the opening of the century, has grown to enormous proportions in the past ten years, so that today there seems to be general agreement that the periodic physical examination of the young child throughout its formative years is desirable.

It is possible, as shown by the study, to coordinate many types of existing health facilities for the medical supervision of large groups of children. This information is especially pertinent just now. Health Centers have been set up by municipal health departments, by social service groups, by hospitals and by independent organizations. They are financed both publicly and privately; they offer every type of preventive care from prenatal supervision, through infant care up to the school period; they may include psychiatric clinics and immunization centers; they may serve as teaching groups to medical students, nurses and to parents, but at one point they all agree—all bring the children of indigent families under the care of trained physicians. The clear presentation of this type of care should stimulate its extension as a ready means of offering immediate protection to a generation of children which, if unguarded, is facing unfathomed difficulties.

MRS. BERTHOLD STRAUSS.

Communicable Disease Control. Report of the Committee on Communicable Disease Control. The White House Conference on Child Health and Protection. The Century Co., New York, 1931. Pp. 243.

The Committee on Communicable Disease Control of the White House Conference numbered twenty-seven individuals who have had wide experience in all phases of communicable disease work, clinical, diagnostic, laboratory, administrative, epidemiologic, and statistical. There has been brought together a great mass of data relating to modern practice in that phase of public health work which deals with contagious diseases, with special emphasis upon the problems of childhood.

This contribution contains valuable statistical tables on the prevalence of communicable diseases in certain areas of the United States as well as a summary of several sickness surveys. The general trend of health department practice in the larger cities is enumerated, especially with respect to control measures for smallpox, typhoid fever, diphtheria, and scarlet fever.

The principal contribution in this volume is the revision of the procedures recommended for the individual diseases, the reporting of which is usually required by the modern health department. The recommendations of the Committee on Communicable Disease Control of the American Public Health Association have been brought up to date and rearranged in light of recent experience. This section of the report is indispensable to the public health administrator and to physicians whose practice pertains to the treatment and prevention of the diseases of childhood whether such physicians be associated with a health department or engaged in private practice. At the close of the volume there are presented definite recommendations for the establishment of well-organized health departments with full-time trained personnel for all local areas in the United States and more especially in the rural districts. Medical participation in continuous programs to secure the protection of children against diphtheria and smallpox is especially recommended. It is hoped that the routine care of the infant by the family physician will involve specific immunization as a routine. The report concludes with recommendations for additional studies and research. The book is well edited and is of particular value because it represents the group judgment of experts who have been engaged in child health work for many years.

HENRY F. VAUGHAN.

Psychology and Psychiatry in Pediatrics: The Problem. The White House Conference on Child Health and Protection. The Century Co., New York. Pp. 166.

The volume on *Psychology and Psychiatry in Pediatrics: The Problem* is an attempt to formulate a point of view which might assist the pediatrician to find his way through these confusing and ill-defined fields of knowledge. The subcommittee found no formal program which they were willing to endorse, but they agreed upon the following statements:

1. The general practitioner, or the pediatrician, is in a position of great natural strength if he chooses to consider the intellectual and emotional factors which cause distress to children and anxiety to parents.

2. On the whole, although pediatricians should be in profitable contact with psychiatrists, psychologists and teachers, there is little evidence that mutual confidence and helpfulness is the rule.

3. The attitude of reserve, of suspicion, or of downright hostility which many pediatricians maintain towards the general group of subjects considered in this report can be explained, in part, by over-eagerness on the part of propagandists who have been tempted to force acceptance of new programs before doctors were convinced of their validity.

This is only a part of the story, however. Many pediatricians have refused to consider the evidence before condemning the general principles which are regarded as valid by almost all workers in psychiatric and psychological enterprises for children.

4. On the whole the committee feels that cooperation of all groups interested in studying the emotional and intellectual difficulties of children will come, but

unless pediatricians make a genuine effort to find their place in the general field of mental hygiene they may well be either disregarded or find that they have sacrificed their present favorable position.

5. It may well be that the psychiatrists and psychologists who have attempted to instruct pediatricians have been unwise in their selection of material or that they have tried to hurry things too much. At present efforts are being made by psychiatrists, individually and in groups, to find the weak places in their plan of teaching.

6. If pediatricians have not profited by the instruction offered it is their privilege to select any attitudes and techniques which they think will be useful. It may well be that a new selection of material will lead to a better method in the hands of the pediatrician.

In general the subcommittee admits that confusion exists but feels entirely convinced that out of the confusion will come relatively simple methods of dealing with many of the important difficulties which face children during their developing years.

BRONSON CROTHER.

Pediatrics: Education and Practice. Report of the Committee on Medical Education. The White House Conference on Child Health and Protection. The Century Company, New York, 1931. 117 pp.

A quotation from the Introduction to this volume points out the reason why the White House Conference on Child Health and Protection was especially interested in the education of the physician: "No matter how little or how fully developed the many types of organizations may be which have to do with the health of the child, in the last analysis, whether they function adequately or not, in fact whether or not most of them exist, depends chiefly upon the help and knowledge of the physician. In organized health work which has so largely occupied the time of the Conference we are apt to forget that *the vast majority of American children today are dependent upon the interest and care of a private physician for their physical well-being and health*, and not upon an organization."

This report comprises the type of study which justifies the recommendations made respecting the necessity for the development of a course in pediatrics that will qualify the physician in general practice to deal effectively with the preventive aspects of pediatrics. It does not attempt to outline a course that would qualify one planning to devote his entire time to the medical care of children. The importance of pediatrics in the field of medicine is stressed very effectively, the fact being emphasized that it deals with the entire child and is really the application of general medicine to a period of life which marks the growth and development of the individual. To acquaint the student with the manifestations peculiar to this age period requires a very different type of instruction from that which is necessary to prepare him to care for adults.

The report is based upon the replies to a series of questionnaires issued to "physicians who specialized in pediatrics"; "physicians who, while not limiting their practice to, were especially interested in, pediatrics," and "physicians in general practice for ten and fifteen years and who practiced in communities of less than 50,000 population." Additional questionnaires were sent to professors of pediatrics in 63 medical schools, to secretaries of state medical societies and to heads of state departments of health (the last two relate specifically to postgraduate instruction). These questionnaires were sent to ascertain the attitude of the physician toward certain phases of pediatric instruction and practice. Only 28 per cent reported their undergraduate instruction as satisfactory.

The following quotation from the report is indicative of the sane procedure that was followed by this committee: "The question of medical education in pediatrics then, from the standpoint from which we are considering it, is a subject with many facets. We must take into consideration the medical school as a whole with the rapid changes and progress that has taken place in recent years. We must think of pediatrics as one of many subjects which must be taught in a limited period of time. We must think of the subject in the light of its scientific development. The place and rôle of pediatrics in the work of the specialist and general practitioner must be considered. The development of public health methods as they apply to children must enter in. Above all we must think of pediatrics as a subject not primarily concerned with the treatment and cure of disease, but as a rather unique medical branch which has to do with the child—an important entity and not a miniature adult. In all this we must bear in mind that the prevention of disease is more important than its cure."

In discussing the report Dr. Alfred Stengel, professor of medicine in the University of Pennsylvania, made the following comment: "This very full report of the committee excites my particular approval because, aside from its comprehensiveness, it exhibits a degree of sanity of judgment and balance of opinion that marks it as somewhat unusual. It stresses the things which are fundamental. It is those things which first of all must gain our attention and secure general approval."

Many detailed tables are given in regard to the scope and extent of pediatric instruction in the medical schools. Among the recommendations of the committee are the extension of postgraduate instruction by state groups. An outline of the scope of a pediatric course is given and 200 teaching hours estimated as the time necessary to cover the material outlined. Recommendations are also made as to the necessary hospital and teaching facilities to teach the subject in a satisfactory manner. Throughout the report stress is placed upon the importance of preventive measures and upon the supervision of growth and development. The report is of interest to all physicians and of special value to those interested in medical education and the teaching of pediatrics.

S. McC. Hamill.

Normal Youth and Its Everyday Problems. Douglas A. Thom, M.D., New York. D. Appleton & Co., 1932, pp. 368.

One of the most difficult questions that comes to the physician is the recommendation of a book on adolescence for the parent or teacher who is struggling with some specific problem. As a matter of fact it is about as difficult for the teacher of medicine to recommend a book to enquiring students and practitioners. While many excellent studies on adolescence have appeared in recent years, most of them have been too general or abstract, or too technically written to be of much service to the average individual. Dr. Thom's new book based on his own experience can be unhesitatingly and unqualifiedly recommended to parents and teachers. Unlike so many books written for parents it is neither trite nor dogmatic.

The book is written from the "case report" standpoint and contains many illustrations from the author's records. These are built up into subjects or chapters, as sex, education, personality, conduct problems, physical development, parental relations, etc. Underlying it all is a thorough knowledge of psychology,

which, however, never intrudes upon the reader's attention by the use of unnecessarily complicated verbiage which psychiatrists and psychologists so often seem to think necessary for expressing (or concealing) their thoughts.

Above all, the book is sane. The reader is conscious throughout not only of a broad sympathetic viewpoint toward youth, but of an unusual understanding on the part of the author. Not only can it be recommended to parents, but it is a book that should be read by every physician who in one way or another has to meet the problems of youth—be it in his own home or practice.

BORDEN S. VEEDER.

Comments

THE discussion that took place at the New Orleans meeting of the Academy in regard to the Jones-Bankhead bill (p. 126, July issue) is indicative of two fundamental points of view. One is that the end in view is more important than the method, the other that the method involved in the bill carries with it such objectionable features that the end in view should be sought for by other methods. We may accept without discussion that every pediatrician, and we hope every physician, is in sympathy with its stated purpose, the reduction of infant and maternal mortality.

Essentially the Jones-Bankhead bill is a continuation of the well-known Sheppard-Towner bill, which aroused such controversy a decade ago. The Sheppard-Towner bill allocated federal funds to the various states for child welfare and maternal health purposes. The states had to match the money dollar for dollar, and the plans for the use of the money in each state had to be approved by a committee of federal officials. A controversy started which waxed fast and furious even after the bill was passed and put into effect. There was no new principle involved in such use of federal funds—it had been and is today being used for agriculture, road building, etc., and is being considered for unemployment relief—except that it involved the medical profession. It was opposed by the American Medical Association on the broad grounds of a step toward "state medicine," and the injection of federal into state affairs. The members of the Section of Diseases of Children of the Association, who had a definite interest in the matter and a knowledge of what the aim of the bill meant to the children of America, were for the most part in favor of the bill. At the 1922 session the Section passed a resolution favoring the bill though the House of Delegates did not sanction the resolution and passed an opposing one.

The Academy of Pediatrics has the function and duty of forming its own opinion on matters relating to the health and welfare of children and of giving expression to its views. We therefore disagree with the view expressed some time ago that the American Medical Association alone has the right to speak for American physicians. With all humility we assert the belief that the opinion of the American Academy of Pediatrics on matters relating to child health and welfare is more likely to be intelligently formed than an opinion on such subjects by any other professional group.

We feel that the action taken by the Academy to lay the matter over was a correct one. This, not because we are either in favor of or opposed to the present bill, but because there was insufficient information presented as to the need for federal aided child and maternal health measures at the present time. So far as the Jones-Bankhead bill itself is concerned we see no reason to oppose it because it is opposed by the American Medical Association or favor it because it is a pet of the League of Women Voters. The attitude of the Academy should be based on certain definite facts which are not difficult to obtain. We can act intelligently upon facts regardless of whether we happen to hold to the principles of Hamilton or Jefferson. The facts are simply these:

Did or did not the Sheppard-Towner bill accomplish what its proponents stated it would?

Were its results of real value and commensurate with the cost?

If it did accomplish valuable results, is there further need at the present time for federal aided child and maternal health activities?

With, and only with, such information can an intelligent opinion be formed and the Academy take a definite stand. The machinery should be put in motion to obtain this information from the pediatricians of the different states. There is a feeling that at the present time an odor of bureaucracy surrounds the whole subject, which is hidden under the worthy object of the bill. The position of the Academy should not be based upon the position taken by any other organization, regardless of whether it is in favor of or opposed to the present bill.

WE ARE interested in presenting two different views of the value of a specific method of therapy in this issue. Both opinions are honest and fairly presented and reflect the viewpoint reached by competent observers. To the practicing physician such conflicting viewpoints may be confusing. The only conclusion that can be drawn, and this reflects the conflicting results of others who have reported on the use of nirvanol in chorea, is that its place is not as yet definitely established in the armamentarium of the practicing physician. There is further need for controlled study and observation in the clinic before its therapeutic value is established.

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